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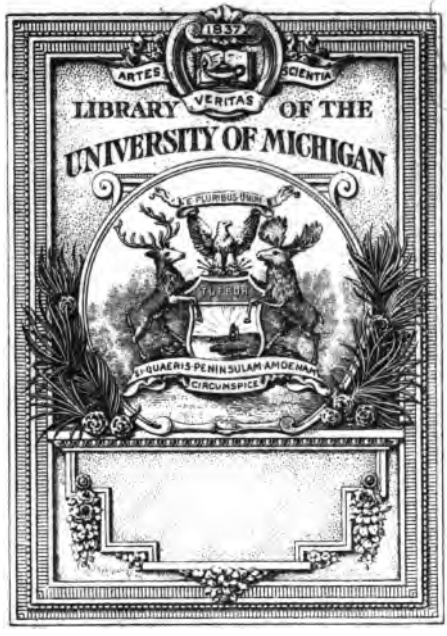
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OCTOBER, 1882.

Those marked thus (†) have paid the Composition Fee in lieu of further annual subscriptions.

Amongst the non-residents those marked thus (*) are entitled by composition to receive the Transactions.

Elected

1846 *ABERCROMBIE, JOHN, M.D., Physician to the Cheltenham General Hospital; 13, Suffolk square, Cheltenham.

1877 ABERCROMBIE, JOHN, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, Charing Cross Hospital; 39, Welbeck street, Cavendish square.

1851 *ACLAND, HENRY WENTWORTH, M.D., F.R.S., Honorary Physician to H.R.H. the Prince of Wales; Regius Professor of Medicine, and Clinical Professor in the University of Oxford.

1847 ACOSTA, ELISHA, M.D., 24, Rue de Luxembourg, St. Honoré, Paris.

Elected

1852 ADAMS, WILLIAM, Surgeon to the Great Northern Hospital and to the National Hospital for the Paralysed and Epileptic; Consulting Surgeon to the National Orthopædic Hospital, Great Portland street; 5, Henrietta street, Cavendish square. C. 1873-4. *Trans.* 3.

1867 AIKIN, CHARLES ARTHUR, 7, Clifton place, Hyde park.

1837 *AINSWORTH, RALPH FAWSETT, M.D., Consulting Physician to the Manchester Royal Infirmary; Cliff Point, Lower Broughton, Manchester.

1839 ALCOCK, SIR RUTHERFORD, K.C.B., K.C.T., K.T.S., D.C.L., late H.M.'s Envoy Extraordinary at the Court of Pekin. *Trans.* 1.

1866 ALLBUTT, THOMAS CLIFFORD, M.A. and M.D., F.R.S., Lecturer on the Practice of Physic at the Leeds School of Medicine, and Physician to the Leeds General Infirmary; 35, Park square, Leeds. *Trans.* 3.

1879 ALLCHIN, WILLIAM HENRY, M.B., Physician to the Westminster Hospital; 5, Chandos street, Cavendish square, W.

1863 ALTHAUS, JULIUS, M.D., Senior Physician to the Hospital for Epilepsy and Paralysis, Regent's park; 36, Bryanston street, Portman square. *Trans.* 2.

1881 ANDERSON, JAMES, A.M., M.D., 84, Wimpole street, Cavendish square.

1862 ANDREW, EDWYN, M.D., Hardwick House, St. John's Hill, Shrewsbury.

1862 ANDREW, JAMES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; 22, Harley street, Cavendish square. S. 1878-9. C. 1881-2. *Trans.* 1.

1820 ANDREWS, THOMAS, M.D., Norfolk, Virginia.

1880 *APPLETON, HENRY, M.D., Staines.

1878 ARNOLD, JOHN, Medical Officer of Health; Trinidad.

1819 †ARNOTT, JAMES MONCRIEFF, F.R.S., Chapel House, Lady Bank, Fifeshire, and 36, Sussex gardens, Hyde park. L. 1826-8. V.P. 1832-3. T. 1835-40. C. 1846, 1855-6. P. 1847-8. *Referee*, 1842-45, 1849-54, 1857-64. *Lib. Com.* 1836. *Trans.* 8.

Elected

1874 AVELING, JAMES H., M.D., Physician to the Chelsea Hospital for Women; 1, Upper Wimpole street, Cavendish square.

1851 *BAKER, ALFRED, Surgeon to the Birmingham General Hospital; 20A, Temple row, Birmingham.

1873 *BAKER, J. WRIGHT, Senior Surgeon to the Derbyshire General Infirmary; 102, Friargate, Derby.

1865 BAKER, WILLIAM MORRANT, Surgeon to, and Lecturer on Physiology at, St. Bartholomew's Hospital; Surgeon to the Evelina Hospital for Sick Children; 26, Wimpole street, Cavendish square. C. 1878-9. *Lib. Com.* 1876-7. *Trans.* 6.

1869 BAKEWELL, ROBERT HALL, M.D., Ross, Westland, New Zealand.

1839 †BALFOUR, THOMAS GRAHAM, M.D., F.R.S., Surgeon General; Coombe Lodge, Wimbledon Park. C. 1852-3. V.P. 1860-1. T. 1872. *Lib. Com.* 1849. *Trans.* 2.

1848 †BALLARD, EDWARD, M.D., Inspector, Medical Department, Local Government Board; 12, Highbury terrace, Islington. C. 1872. V.P. 1875-6. *Referee*, 1853-71. *Lib. Com.* 1855. *Trans.* 5.

1866 *BANKS, JOHN THOMAS, M.D., Physician in Ordinary to the Queen in Ireland; Physician to Richmond, Whitworth, and Hardwicke Hospitals; Consulting Physician to the Coombe Hospital; Member of the Senate of the Queen's University in Ireland; 11, Merrion square east, Dublin.

1847 †BARCLAY, ANDREW WHYTE, M.D., Consulting Physician to St. George's Hospital; Medical Officer of Health for Chelsea; Whitney Wood, Stevenage. S. 1857-60. L. 1861-2. C. 1865-6. V.P. 1872-3. P. 1881. *Referee*, 1863-4, 1870-1, 1877-80. *Trans.* 2.

1879 BARKER, ARTHUR EDWARD JAMES, Assistant Surgeon to, and Assistant Professor of Clinical Surgery at, University College Hospital; 87, Harley street, Cavendish square. *Trans.* 2.

Elected

1882 BARKER, FREDERICK CHARLES, M.D., Surgeon-Major, Bombay Medical Service [care of ARTHUR E. J. BARKER, 87, Harley street].

1833 †BARKER, THOMAS ALFRED, M.D., Consulting Physician to St. Thomas's Hospital; 27, Wimpole street. C. 1844-5. V.P. 1853-4. T. 1860-2. *Referee*, 1848-51. *Trans.* 6.

1876 BARLOW, THOMAS, M.D. and B.S. Lond., Assistant Physician to University College Hospital, and to the Hospital for Sick Children, Great Ormond Street; 10, Montague street, Russell square.

1881 *BARNES, HENRY, M.D., F.R.S. Ed., Physician to the Cumberland Infirmary; 6, Portland square, Carlisle.

1861 BARNES, ROBERT, M.D., Obstetric Physician to, and Lecturer on Midwifery at, St. George's Hospital; 15, Harley street, Cavendish square. C. 1877-8. *Referee*, 1867-76. *Lib. Com.* 1869-73. *Trans.* 4.

1864 BARRATT, JOSEPH GILLMAN, M.D., 8, Cleveland gardens, Bayswater.

1880 BARROW, A. BOYCE, Pathological Registrar, King's College Hospital; 17, Welbeck street, Cavendish square, W.

1840 BARROW, BENJAMIN, Surgeon to the Royal Isle of Wight Infirmary; Southlands, Ryde, Isle of Wight.

1859 BARWELL, RICHARD, Surgeon to, and Lecturer on Surgery at, the Charing Cross Hospital; 32, George street, Hanover square. C. 1876-77. *Referee*, 1868-75, 1879-82. *Trans.* 8.

1868 BASTIAN, HENRY CHARLTON, M.A., M.D., F.R.S., Professor of Pathological Anatomy in University College, London, and Physician to University College Hospital; 20, Queen Anne street, Cavendish square. *Trans.* 1.

1874 BAXTER, EVAN BUCHANAN, M.D., Professor of Materia Medica at King's College, London; Physician to King's College Hospital; Examiner in Materia Medica at the University of London; 28, Weymouth street, Portland place. *Referee*, 1881–2.

1875 BEACH, FLETCHER, M.B., Medical Superintendent, Metropolitan District Asylum, Darenth, near Dartford, Kent.

Elected

1862 BEALE, LIONEL SMITH, M.B., F.R.S., Professor of the Principles and Practice of Medicine in King's College, London, and Physician to King's College Hospital; 61, Grosvenor street. C. 1876-77. *Referee*, 1873-5. *Trans.* 1.

1860 *BEALEY, ADAM, M.D., M.A. Camb., Oak Lea, Harrogate.

1856 BEARDSLEY, AMOS, F.L.S., Bay villa, Grange-over-Sands, Lancashire.

1871 BECK, MARCUS, M.S., Teacher of Operative Surgery, and Assistant Professor of Clinical Surgery in University College, London; and Assistant Surgeon to University College Hospital; 30, Wimpole street, Cavendish square. *Referee*, 1882. *Lib. Com.* 1881–2.

1880 BEEVOR, CHARLES EDWARD, M.D.; 129, Harley street, Cavendish square.

1858 BEGLEY, WILLIAM CHAPMAN, A.M., M.D., late of the Middlesex County Lunatic Asylum, Hanwell; 26, Saint Peter's square, Hammersmith. C. 1877–8.

1871 BELLAMY, EDWARD, Surgeon to, and Lecturer on Anatomy at, Charing Cross Hospital; Professor of Anatomy in the Science and Art Department, South Kensington; 17, Wimpole street, Cavendish square. *Referee*, 1882. *Lib. Com.* 1879-81.

1847 BENNET, JAMES HENRY, M.D., The Ferns, Weybridge, and Mentone.

1880 BENNETT, ALEX. HUGHES, M.D., Assistant Physician to the Westminster Hospital; 13, Old Cavendish street.

1877 BENNETT, WILLIAM HENRY, Assistant Surgeon to, and Lecturer on Anatomy at, St. George's Hospital; Surgeon to the Belgrave Hospital for Children; 4, Chesterfield street, Mayfair.

1845 †BERRY, EDWARD UNWIN, 76, Gower street, Bedford square.

1820 BERTIN, STEPHEN, Paris.

1872 BEVERLEY, MICHAEL, M.D., Assistant Surgeon to the Norfolk and Norwich Hospital; 63, St. Giles's street, Norwich.

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1865 *BICKERSTETH, EDWARD ROBERT, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Clinical Surgery in the Liverpool Royal Infirmary School of Medicine; 2, Rodney street, Liverpool. *Trans.* 1.

1882 BINDLEY, PHILIP HENRY, M.B., Watford.

1878 BINDON, WILLIAM JOHN VEREKER, M.D., Appin, West End lane, West Hampstead.

1854 BIRD, PETER HINCKES, F.L.S.

1856 BIRD, WILLIAM, Consulting Surgeon to the West London Hospital; Bute House, Hammersmith.

1849 †BIRKETT, EDMUND LLOYD, M.D., Consulting Physician to the City of London Hospital for Diseases of the Chest; 48, Russell square. C. 1865-6. *Referee*, 1851-9.

1851 †BIRKETT, JOHN, F.L.S., Consulting Surgeon to Guy's Hospital; Corresponding Member of the "Société de Chirurgie" of Paris; Inspector of Anatomy for the Provinces in England and Wales; 59, Green street, Grosvenor square. L. 1856-7. S. 1863-5. C. 1867-8. T. 1870-78. V.P. 1879-80. *Referee*, 1851-5, 1866, 1869, *Sci. Com.* 1863. *Lib. Com.* 1852. *Trans.* 8.

1866 BISHOP, EDWARD, M.D., Cintra park, Upper Norwood.

1881 BISS, CECIL YATES, M.B., Assistant Physician to, and Lecturer on Botany at, the Middlesex Hospital; Assistant Physician to the Hospital for Consumption; 2, Old Burlington street, and Claremont, Sydenham park, S.E.

1865 BLANCHET, HILARION, Examiner to the College of Physicians and Surgeons, Lower Canada; 6, Palace street, Quebec, Canada east.

1865 BLANDFORD, GEORGE FIELDING, M.D., Lecturer on Psychological Medicine at St. George's Hospital; 71, Grosvenor street.

1867 BLOXAM, JOHN ASTLEY, Surgeon to, and Teacher of Operative Surgery in, Charing Cross Hospital; Surgeon for Out-Patients to the Lock Hospital; Junior Surgeon to the West London Hospital; 8, George street, Hanover square.

1823 BOJANUS, LOUIS HENRY, M.D., Wilna.

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1846 †Bostock, John Ashton, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-Major, Scots Fusilier Guards; 73, Onslow gardens, Brompton. C.1861-2. V.P. 1870-71. *Sci. Com.* 1867.

1869 Bourne, Walter, M.D. [care of the National Bank of India, 80, King William street, City.]

1882 Bowlby, Anthony A., Curator of the Museum, St. Bartholomew's Hospital, 75, Warrington crescent, W.

1870 *Bowles, Robert Leamon, M.D., 8, West terrace, Folkestone.

1841 †Bowman, William, LL.D., F.R.S., F.L.S., Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 5, Clifford street, Bond street. C. 1852-3. V.P. 1862. *Referee*, 1845-50, 1854-6. *Lib. Com.* 1847. *Trans.* 3.

1862 Brace, William Henry, M.D., 7, Queen's Gate terrace, Kensington.

1874 Bradshaw, A. F., Surgeon-Major; Surgeon to the Rt. Hon. the Commander in Chief in India; Army Head Quarters, Bengal Presidency. [Agent: Vesey W. Holt, 17, Whitehall place.]

1867 *Brett, Alfred T., M.D., Watford, Herts.

1876 Bridges, Robert, M.B.

1867 Bridgewater, Thomas, M.B. Lond., Harrow-on-the-Hill, Middlesex.

1868 Broadbent, William Henry, M.D., Physician to, and Lecturer on Medicine at, St. Mary's Hospital; Consulting Physician to the London Fever Hospital; 34, Seymour street, Portman square. *Referee*, 1881–2. *Trans.* 3.

1851 †Brodhurst, Bernard Edward, F.L.S., Surgeon to the Royal Orthopædic Hospital; 20, Grosvenor street. C. 1868-9. *Lib. Com.* 1862-3. *Trans.* 2. *Pro.* 1.

1872 Brodie, George Bernard, M.D., Consulting Physician-Accoucheur to Queen Charlotte's Hospital; 3, Chesterfield street, Mayfair. *Trans.* 1.

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1860 BROWN-SÉQUARD, CHARLES EDOUARD, M.D., LL.D., F.R.S., Laureate of the Academy of Sciences of Paris; Professor of Medicine at the College of France; Professor of General Physiology at the Museum of Natural History; Paris. *Sci. Com.* 1862.

1878 BROWNE, JAMES CRICHTON, M.D., LL.D., F.R.S.Ed., Lord Chancellor's Visitor in Lunacy; 7, Cumberland Terrace, Regent's Park.

1880 BROWNE, JAMES WILLIAM, M.B., 8, Norland place, Uxbridge road.

1881 BROWNE, JOHN WALTON, M.D., Surgeon to the Belfast Ophthalmological Hospital; 10, College square N., Belfast.

1881 BROWNE, OSWALD A., M.A., St. Bartholomew's Hospital; 25, Bernard Street, Russell Square.

1874 BRUCE, JOHN MITCHELL, M.D., Physician to, and Lecturer on Materia Medica at, the Charing Cross Hospital; Assistant Physician to the Hospital for Consumption, Brompton; 70, Harley street. *Trans.* 1.

1871 BRUNTON, THOMAS LAUDER, M.D., F.R.S., Assistant Physician to, and Lecturer on Materia Medica at, St. Bartholomew's Hospital; 50, Welbeck street, Cavendish square. *Referee*, 1880-82. *Lib. Com.* 1882.

1860 BRYANT, THOMAS, Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 53, Upper Brook street, Grosvenor square. C. 1873-4. *Sci. Com.* 1863. *Referee*, 1882. *Lib. Com.* 1868-71. *Trans.* 9. *Pro.* 1.

1855 BRYANT, WALTER JOHN, M.R.C.P. Edinb.; Physician to the Home for Incurable Children, Maida vale; 23A, Sussex square, Hyde park gardens.

1823 BUCHANAN, B. BARTLET, M.D.

1864 BUCHANAN, GEORGE, M.D., F.R.S., Medical Officer of the Local Government Board; Member of the Senate of the University of London; 24, Nottingham place, Marylebone road.

1864 BUCKLE, FLEETWOOD, M.D.

1876 BUCKNILL, JOHN CHARLES, M.D., F.R.S.; E 2, Albany, Piccadilly, and Hill Morton Hall, Rugby,

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1881 BULLER, AUDLEY CECIL, St. Bartholomew's Hospital.

1833 †BURROWS, SIR GEORGE, Bart., M.D., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen; Consulting Physician to St. Bartholomew's Hospital; Member of the Senate of the University of London; 18, Cavendish square. C. 1839-40, 1858-9. T. 1845-7. V. P. 1849-50. P. 1869-70. *Referee*, 1842-6, 1850-7, 1861-68, 1875-81. *Lib. Com.* 1836. *Trans.* 2.

1837 †BUSK, GEORGE, F.R.S., F.L.S., Consulting Surgeon to the Seamen's Hospital, Greenwich; Member of the Senate of the University of London; 32, Harley street, Cavendish square. C. 1847-8. V.P. 1855. T. 1866. *Referee*, 1846-54, 1857-65. *Lib. Com.* 1847. *Trans.* 4.

1873 BUTLIN, HENRY TRENTHAM, Assistant Surgeon to, and Demonstrator of Practical Surgery at, St. Bartholomew's Hospital; Assistant Surgeon to the West London Hospital; 47, Queen Anne street, Cavendish square. *Trans.* 3.

1871 BUTT, WILLIAM F., 25, Park street, Park lane.

1868 BUZZARD, THOMAS, M.D., Physician to the National Hospital for the Paralysed and Epileptic; 56, Grosvenor street, Grosvenor square.

1851 *CADGE, WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 24, St. Giles's street, Norwich. *Trans.* 1.

1875 CARTER, CHARLES HENRY, M.D., Physician to the Hospital for Women, Soho square; 45, Great Cumberland place, Hyde park.

1853 CARTER, ROBERT BRUDENELL, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. George's Hospital; 69, Wimpole street, Cavendish square. *Trans.* 1.

1845 †CARTWRIGHT, SAMUEL, late Professor of Dental Surgery at King's College, London, and Surgeon-Dentist to King's College Hospital; Consulting Surgeon to the Dental Hospital; 32, Old Burlington street. C. 1860-1. *Sci. Com.* 1863.

Elected

1879 CARTWRIGHT, S. HAMILTON, Professor of Dental Surgery at King's College; 32, Old Burlington street.

1868 CAVAFY, JOHN, M.D., Physician to St. George's Hospital; 2, Upper Berkeley street, Portman square. *Trans.* 1.

1871 CAYLEY, WILLIAM, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, the Middlesex Hospital; Physician to the London Fever Hospital and to the North-Eastern Hospital for Children; 58, Welbeck street, Cavendish square.

1845 †CHALK, WILLIAM OLIVER, 3, Nottingham terrace, York gate, Regent's park. C. 1872-3.

1844 †CHAMBERS, THOMAS KING, M.D., Hon. Physician to H.R.H. the Prince of Wales; Consulting Physician to St. Mary's Hospital; Consulting Physician to the Lock Hospital; Shrubs Hill House, Sunningdale, Staines. C. 1861. V.P. 1867. L. 1869-72. *Referee*, 1851-60, 1866. *Lib. Com.* 1852, 1868. *Trans.* 1.

1879 CHAMPNEYS, FRANCIS HENRY, M.A., M.B., Assistant Obstetric Physician to St. George's Hospital; 60, Great Cumberland place *Trans.* 3.

1859 CHANCE, FRANK, M.D., Burleigh House, Sydenham Hill.

1849 CHAPMAN, FREDERICK, Old Friars, Richmond Green, Surrey.

1877 CHARLES, T. CRANSTOUN, M.D., Lecturer on Practical Physiology at St. Thomas's Hospital; 25, The Terrace, Barnes.

1881 *CHAVASSE, THOMAS FREDERICK, M.D., C.M., Surgeon to the General Hospital, Birmingham; 108, New Hall street, Birmingham.

1868 CHEADLE, WALTER BUTLER, M.D., Physician (with charge of Out-patients) to, and Lecturer on Medicine at, St. Mary's Hospital; Physician to the Hospital for Sick Children; 2, Hyde park place, Cumberland gate.

Elected

1879 CHEYNE, WILLIAM WATSON, M.B., Assistant Surgeon and Demonstrator of Surgery to King's College Hospital; 14, Mandeville place, Manchester square, W.

1873 *CHISHOLM, EDWIN, M.D., Abergeldie, Ashfield, near Sydney, New South Wales.

1865 CHOLMELEY, WILLIAM, M.D., Physician to the Great Northern Hospital, and to the Margaret Street Infirmary for Consumption; 63, Grosvenor street, Grosvenor square. C. 1881-2. *Referee*, 1873-80.

1872 CHRISTIE, THOMAS BEITH, M.D., Medical Superintendent, Royal India Asylum, Ealing.

1866 CHURCH, WILLIAM SELBY, M.D., Physician to, and Lecturer on Clinical Medicine at, St. Bartholomew's Hospital; 130, Harley street, Cavendish square. *Referee* 1874-81.

1860 CLARK, ANDREW, M.D., LL.D., Aberd., Physician to, and Lecturer on Medicine at, the London Hospital; 16, Cavendish square. C. 1875.

1879 CLARK, ANDREW, Assistant Surgeon to, and Lecturer on Practical Surgery at, the Middlesex Hospital; 19, Cavendish place, Cavendish square, W.

1839 †CLARK, FREDERICK LE GROS, F.R.S., Consulting Surgeon to St. Thomas's Hospital; The Thorns, Sevenoaks. S. 1847-9. V.P. 1855-6. *Referee*, 1859-81. *Lib. Com.* 1847. *Trans.* 5.

1882 CLARKE, ERNEST, Assistant Chloroformist to St. Bartholomew's Hospital, 31, Belsize Avenue, Hampstead.

1848 †CLARKE, JOHN, M.D., 42, Hertford street, May Fair. C. 1866.

1881 CLARKE, W. BRUCE, M.B., Demonstrator of Anatomy at St. Bartholomew's Hospital; 46, Harley street, Cavendish square.

1866 CLARKE, WILLIAM FAIRLIE, M.D., M.A. Oxon., Southborough, Tunbridge Wells. *Trans.* 2.

Elected

1842 †Clayton, Oscar Moore Passey, Extra Surgeon-in-Ordinary to H.R.H. the Prince of Wales, and Surgeon-in-Ordinary to H.R.H. the Duke of Edinburgh; 5, Harley street, Cavendish square. C. 1865.

1879 Clutton, Henry Hugh, M.A., M.B., Assistant Surgeon to, and Lecturer on Forensic Medicine at, St. Thomas's Hospital; 77, Lambeth Palace road.

1857 Coates, Charles, M.D., Physician to the Bath General and Royal United Hospitals; 10, Circus, Bath.

1868 Cockle, John, M.D., F.L.S., Physician to the Royal Free Hospital; 13, Spring gardens, Charing cross. *Trans.* 2.

1865 Cooper, Alfred, Surgeon to the Lock Hospital; Assistan Surgeon to St. Mark's Hospital; Surgeon to the West London Hospital; 9, Henrietta street, Cavendish square.

1843 †Cooper, William White, Surgeon-Oculist in Ordinary to H.M. the Queen; Consulting Ophthalmic Surgeon to St. Mary's Hospital; 19, Berkeley square. C. 1858-9. V.P. 1873-4. *Lib. Com.* 1847, 1856-7.

1868 Cornish, William Robert, Surgeon-Major, Madras Army; Sanitary Commissioner for Madras; Secretary to the Inspector-General, Indian Medical Department.

1860 *Corry, Thomas Charles Steuart, M.D., Surgeon to the Belfast General Dispensary; 146, Donegall Pass, Belfast.

1853 Cory, William Gillett, M.D., 1, Caledonia place, Clifton, Bristol.

1864 Coulson, Walter John, Surgeon to the Lock Hospital, 17, Harley street, Cavendish square.

1860 †Couper, John, Surgeon to the London Hospital; Assistant Surgeon to the Royal London Ophthalmic Hospital; 80, Grosvenor street. C. 1876. *Referee* 1882.

1877 Coupland, Sidney, M.D., Physician to, and Lecturer on Pathological Anatomy at, the Middlesex Hospital; 14 Weymouth street, Portland place.

Elected

1862 COWELL, GEORGE, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon to the Royal Westminster Ophthalmic Hospital; Surgeon to the Victoria Hospital for Children; 19, George street, Hanover square. C. 1882.

1841 CRAWFORD, MERVYN ARCHDALL NOTT, M.D., Millwood, Wilbury road, Brighton. C. 1853-4.

1868 CRAWFORD, THOMAS, M.D., Director General, Army Medical Department; 6, Whitehall yard.

1873 CREIGHTON, CHARLES, M.D., 25, Savile row. *Referee*, 1882. *Trans.* 1.

1869 *CRESSWELL, PEARSON R., Dowlais, Merthyr Tydvil.

1874 CRIPPS, WILLIAM HARRISON, Assistant Surgeon to St. Bartholomew's Hospital; Surgeon to the Great Northern Hospital; Assistant Surgeon to the Royal Free Hospital; 6, Stratford place, Oxford street. *Trans.* 1.

1847 †CRITCHETT, GEORGE, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 21, Harley street, Cavendish square. C. 1865. V.P. 1872. *Referee*, 1867-71. *Trans.* 1.

1868 CROFT, JOHN, Surgeon to, and Lecturer on Clinical Surgery at, St. Thomas's Hospital; 48, Brook street, Grosvenor square. *Lib. Com.* 1877-8. *Trans.* 1.

1862 CROMPTON, SAMUEL, M.D., late Physician to the Salford Royal Hospital and Dispensary; Brookmead, Cranleigh, Surrey.

1837 CROOKES, JOHN FARRAR, 45, Augusta gardens, Folkestone.

1860 CROSS, RICHARD, M.D., Carlton House, Belmont road, Scarborough.

1872 CROSSE, THOMAS WILLIAM, Surgeon to the Norfolk and Norwich Hospital; 22, St. Giles's street, Norwich.

1849 *CROWFOOT, WILLIAM EDWARD, Beccles, Suffolk.

1879 CUMBERBATCH, A. ELKIN, Demonstrator of Anatomy at St. Bartholomew's Hospital; 17, Queen Anne street.

1846 CURLING, HENRY, Consulting Surgeon to the Margate Royal Sea-Bathing Infirmary, and the Ramsgate Seamen's Infirmary; Ramsgate, Kent.

Elected

1837 †CURLING, THOMAS BLIZARD, F.R.S., Consulting-Surgeon to the London Hospital; 27, Brunswick square, Brighton. S. 1845-6. C. 1850. T. 1854-7. V.P. 1859. P. 1871-2. *Referee*, 1844-6, 1851-3, 1858, 1865-70, 1875-9. *Sci. Com.* 1863. *Lib. Com.* 1839. *Trans.* 13. *Pro.* 1.

1873 CURNOW, JOHN, M.D., Professor of Anatomy at King's College, London, and Physician to King's College Hospital; 3, George street, Hanover square.

1847 CURREY, JOHN EDMUND, M.D., Lismore, County Waterford.

1822 CUSACK, CHRISTOPHER JOHN, Chateau d'Eu, France.

1872 DALBY, WILLIAM BARTLETT, M.B., Lecturer on Aural Surgery at St. George's Hospital; 18, Savile row. *Trans.* 2.

1836 *DANIEL, JAMES STOCK, Ramsgate, Kent.

1877 DARBISHIRE, SAMUEL DUKINFIELD, M.B., Physician to the Radcliffe Infirmary, Oxford.

1879 DARWIN, FRANCIS, M.B., F.R.S., Down, Bromley, Kent.

1848 DAUBENY, HENRY, M.D., San Remo, Italy.

1874 DAVIDSON, ALEXANDER, M.D., Physician to the Liverpool Northern Hospital; 49, Rodney street, Liverpool.

1853 DAVIES, ROBERT COKER NASH, Rye, Sussex.

1852 DAVIES, WILLIAM, M.D., 18, Gay street, Bath.

1876 DAVIES-COLLEY, J. NEVILLE C., M.C., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; 36, Harley street, Cavendish square. *Trans.* 2.

1878 DAVY, RICHARD, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; 33, Welbeck street, Cavendish square.

1882 DAWSON, YELVERTON, M.D., 28, Hyde park street.

1867 DAY, WILLIAM HENRY, M.D., Physician to the Samaritan Free Hospital for Women and Children; 10, Manchester square.

1878 DENT, CLINTON THOMAS, Assistant Surgeon to St. George's Hospital; 19, Savile row, Burlington gardens. *Trans.* 2.

Elected

1846 *DENTON, SAMUEL BEST, M.D., Ivy Lodge, Hornsea, Hull.

1859 †DICKINSON, WILLIAM HOWSHIP, M.D., Physician to, and Lecturer on Medicine at St. George's Hospital, and Senior Physician to the Hospital for Sick Children, Examiner in Medicine at the University of London; 9, Chesterfield street, Mayfair. C. 1874-5. *Referee*, 1869-73, 1882. *Sci. Com*, 1867-79 *Trans.* 13.

1839 †DIXON, JAMES, Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Consulting Ophthalmic Surgeon to the Asylum for Idiots; Harrow Lands, Dorking. L. 1849-55. V.P. 1857-8. T. 1863-4. C. 1866-7. *Referee*, 1865. *Lib. Com.* 1845-8. *Trans.* 4.

1862 DOBELL, HORACE B., M.D., Consulting Physician to the Royal Hospital for Diseases of the Chest, City road; Streate place, Bournemouth. *Trans.* 2.

1845 DODD, JOHN.

1879 DONKIN, HORATIO, M.B., 60, Upper Berkeley street, Portman square.

1877 DORAN, ALBAN HENRY GRIFFITHS, Surgeon to Out-Patients, Samaritan Hospital; Pathological Assistant to the Museum of the Royal College of Surgeons of England; 51, Seymour street, Portman square.

1863 DOWN, JOHN LANGDON HAYDON, M.D., Physician to, and Lecturer on Clinical Medicine at, the London Hospital; 81, Harley street, Cavendish square. C. 1880. *Trans.* 2.

1867 DRAGE, CHARLES, M.D., Hatfield, Herts.

1879 DREWITT, F. G. DAWTREY, M.B. Ox., Savile Club, 15, Savile row, and 52, Brook street, Grosvenor square.

1853 DRUITT, ROBERT, F.R.C.P. [8, Strathmore gardens, Kensington mall.] *Trans.* 2.

1880 DRURY, CHARLES DENNIS HILL, M.D., Bondgate, Darlington.

1865 DRYSDALE, CHARLES ROBERT, M.D., Physician to the Farringdon Dispensary; Assistant-Physician to the Metropolitan Free Hospital; 65, Regent street, W.

Elected

1865 DUCKWORTH, DYCE, M.D., Assistant-Physician to, and Lecturer on Skin Diseases at, St. Bartholomew's Hospital; 11, Grafton street, Bond street. *Trans.* 1.

1876 DUDLEY, WILLIAM LEWIS, M.D., Physician to the City Dispensary; 125, Cromwell road, South Kensington.

1845 DUFF, GEORGE, M.D., High street, Elgin.

1874 DUFFIN, ALFRED BAYNARD, M.D., Professor of Pathological Anatomy in King's College, London, and Physician to King's College Hospital; 18, Devonshire street, Portland place.

1874 DUKA, THEODORE, M.D., [Surgeon-Major, H.M.'s Bengal Army]; Torquay, Devonshire.

1871 DUKE, BENJAMIN, 1, Cavendish terrace, Clapham common.

1871 *DUKES, CLEMENT, M.D. and B.S., Horton crescent, Rugby, Warwickshire.

1867 DUKES, M. CHARLES, M.D., Wellesley Villa, Wellesley road, Croydon.

1880 DUNBAR, JAMES JOHN MACWHIRTER, M.D., Argyle House, Clapham common.

1877 DUNCAN, JAMES MATTHEWS, M.D., LL.D. Ed., F.R.S. Ed., Obstetric Physician to, and Lecturer on Midwifery and Diseases of Women at, St. Bartholomew's Hospital; Examiner in Obstetric Medicine, University of London; 71, Brook street, Grosvenor square. *Referee*, 1881–2.

1863 DURHAM, ARTHUR EDWARD, F.L.S., Surgeon to, and Lecturer on Surgery at, Guy's Hospital; 82, Brook street, Grosvenor square. C. 1876-7. *Referee*, 1880-1. *Sci. Com.* 1867. *Lib. Com.* 1872-5. *Trans.* 5.

1874 DURHAM, FREDERIC, M.B., Surgical Registrar to Guy's Hospital; 38, Brook street, Grosvenor square.

1843 DURRANT, CHRISTOPHER MERCER, M.D., Physician to the East Suffolk and Ipswich Hospital; Ipswich, Suffolk.

1872 EAGER, REGINALD, M.D., Northwoods, near Bristol.

Elected

1836 EARLE, JAMES WILLIAM, late of Norwich.

1868 EASTES, GEORGE, M.B. Lond., Surgeon-Accoucheur to the Western General Dispensary; 69, Connaught street, Hyde park square.

1824 EDWARDS, GEORGE.

1823 EGERTON, CHARLES CHANDLER, Kendall Lodge, Epping.

1869 ELAM, CHARLES, M.D., Assistant-Physician to the National Hospital for the Paralysed and Epileptic; 75, Harley street, Cavendish square.

1861 *ELLIOT, ROBERT, M.D., Physician to the Fever Hospital and to the Dispensary, Carlisle; Coroner for Carlisle; 35, Lowther street, Carlisle.

1848 ELLIS, GEORGE VINER, late Professor of Anatomy in University College, London; Minsterworth, Gloucester. C. 1863-4. *Trans.* 2.

1868 ELLIS, JAMES, M.D., the Sanatorium, Anaheim, Los Angeles County, California.

1854 *ELLISON, JAMES, M.D., Surgeon-in-Ordinary to the Royal Household, Windsor; 14, High street, Windsor.

1842 †ERICHSEN, JOHN ERIC, F.R.S., Surgeon Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery in University College, London, and Consulting Surgeon to University College Hospital; 6, Cavendish place, Cavendish sq. C. 1855-6. V.P. 1868. P. 1879-80. *Referee*, 1866-7. *Lib. Com.* 1844-7, 1854. *Trans.* 2.

1874 EVANS, GEORGE HENRY, M.D.

1879 EVE, FREDERIC S., Curator of the Museum, St. Bartholomew's Hospital; 14, Furnival's Inn, Holborn. *Trans.* 2.

1877 EWART, WILLIAM, M.B., Assistant Physician to St. George's Hospital; Lecturer on Physiological Chemistry at St. George's Hospital; 33, Curzon street, Mayfair.

1875 *FAGAN, JOHN, Surgeon to the Belfast Hospital for Sick Children; 11, College square north, Belfast.

Elected

1864 †FAGGE, CHARLES HILTON, M.D., Physician to, and Lecturer on Pathology at, Guy's Hospital; Examiner in Medicine at the University of London; 76, Grosvenor street. C. 1880-81. *Referee*, 1874-9. *Sci. Com.* 1879. *Lib. Com.* 1875-9. *Trans.* 7.

1869 FAIRBANK, FREDERICK ROYSTON, M.D., 46, Hallgate, Doncaster.

1862 FARQUHARSON, ROBERT, M.D., M.P. *Lib. Com.* 1876-80.

1844 †FARRE, ARTHUR, M.D., F.R.S., Physician Extraordinary to H.M. the Queen; Physician-Accoucheur to H.R.H. the Princess of Wales; 18, Albert Mansions, Victoria street, Westminster. C. 1857. V.P. 1864. *Referee*, 1848-54, 1861-3, 1865-6. *Sci. Com.* 1863. *Lib. Com.* 1847.

1872 FAYRER, SIR JOSEPH, K.C.S.I., M.D., F.R.S., Honorary Physician to H.M. the Queen, and to H.R.H. the Prince of Wales, and Physician to H.R.H. the Duke of Edinburgh; Surgeon-General, late Bengal Medical Service; Examining Medical Officer to the Secretary of State for India in Council; President of the Indian Medical Board; 53, Wimpole street, Cavendish square. *Referee*, 1881–2.

1872 *FENWICK, JOHN C. J., M.D., Physician to the Durham County Hospital; 16, Old Elvet, Durham.

1863 FENWICK, SAMUEL, M.D., Physician to the London Hospital; 29, Harley street, Cavendish square. C. 1880. *Referee*, 1882. *Trans.* 4.

1880 FERRIER, DAVID, M.D., F.R.S., Professor of Forensic Medicine at King's College, London, and Physician to King's College Hospital; Physician for Out-patients to the National Hospital for the Paralysed and Epileptic; 16, Upper Berkeley street, Portman square, W.

1852 *FIELD, ALFRED GEORGE.

1849 †FINCHAM, GEORGE TUPMAN, M.D., Physician to, and Joint Lecturer on Medicine at, the Westminster Hospital; 13, Belgrave road, Pimlico. C. 1871.

Elected

1879 FINLAY, DAVID WHITE, M.D., Assistant Physician to, and Lecturer on Forensic Medicine at, the Middlesex Hospital; Physician to the Royal Hospital for Diseases of the Chest; 21, Montagu street, Portman square.

1866 FISH, JOHN CROCKETT, B.A., M.D. Camb., Assistant Physician to the West London Hospital; 92, Wimpole street, Cavendish square.

1866 FITZPATRICK, THOMAS, M.D., M.A., Dublin; Physician to the Western General Dispensary; 30, Sussex gardens, Hyde park.

1842 FLETCHER, THOMAS BELL ELCOCK, M.D., Consulting Physician to the Birmingham General Hospital; 43, Clarendon square, Leamington. *Trans.* 1.

1864 *FOLKER, WILLIAM HENRY, Surgeon to the North Staffordshire Infirmary; Bedford House, Hanley, Staffordshire.

1877 FONMARTIN, HENRY DE, M.D., Knaphill, Woking, Surrey.

1848 †FORBES, JOHN GREGORY, Egerton House, Egerton, Ashford, Kent. C. 1868-9. *Lib. Com.* 1855. *Trans.* 2.

1852 †FORSTER, JOHN COOPER, *Treasurer*, Consulting Surgeon to the Royal Hospital for Children and Women; Vice-President of the Royal College of Surgeons; 29, Upper Grosvenor street. C. 1868-9. S. 1873-5. V.P. 1877-8. T. 1879-82. *Referee*, 1870-2, 1876. *Pro.* 1.

1877 *FORTESCUE, GEORGE, M.B., late Surgeon to the Sydney Infirmary; 6, Lyons terrace, Sydney, New South Wales.

1865 FOSTER, BALTHAZAR WALTER, M.D., Professor of Medicine at the Queen's College, Birmingham, and Physician to the Birmingham General Hospital; 16, Temple row, Birmingham.

1859 FOX, EDWARD LONG, M.D., Consulting Physician to the Bristol Royal Infirmary, and Lecturer on Medicine at the Bristol School of Medicine; Church House, Clifton, Gloucestershire.

1880 FOX, THOMAS COLCOTT, B.A., M.B., Physician to the St. George's and St. James's Dispensary; 14, Harley street, Cavendish square.

Elected

1858 Fox, Wilson, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Physician in Ordinary to H.R.H. the Duke of Edinburgh; Holme Professor of Clinical Medicine in University College, London, and Physician to University College Hospital; 67, Grosvenor street. C. 1875-6. *Referee*, 1869-74. *Lib. Com.* 1866-70, 1874. *Trans.* 3.

1871 Frank, Philip, M.D., Cannes, France.

1843 Fraser, Patrick, M.D. C. 1866.

1868 Freeman, William Henry, 21, St. George's square, South Belgravia.

1836 †French, John George, 10, Cunningham place, Maida hill. C. 1852-3.

1876 Furner, Willoughby, 111, King's road, Brighton.

1864 *Gairdner, William Tennant, M.D., Physician in Ordinary to H.M. the Queen in Scotland; Professor of the Practice of Medicine in the University of Glasgow; Physician to the Glasgow Royal Infirmary; 225, St. Vincent street, Glasgow.

1874 Galabin, Alfred Lewis, M.A., M.D., Assistant Obstetric Physician to, and Lecturer on Midwifery and the Diseases of Women at, Guy's Hospital; Assistant-Physician to the Hospital for Sick Children; 14, St. Thomas's street, Southwark. *Referee*, 1882. *Trans.* 2.

1865 Gant, Frederick James, Senior Surgeon to the Royal Free Hospital; 16, Connaught square, Hyde park. C. 1880-81. *Lib. Com.* 1882. *Trans.* 3.

1867 Garland, Edward Charles, L.R.C.P. Edin., Yeovil, Somerset.

1867 Garlike, Thomas W., Malvern Villa, Churchfield road, Ealing.

1854 Garrod, Alfred Baring, M.D., F.R.S., Consulting Physician to King's College Hospital; 10, Harley street, Cavendish square. C. 1867. V.P. 1880-81. *Referee*, 1855-65. *Trans.* 8.

1879 Garstang, Thomas Walter Harropp, Oakleigh, Dobcross, near Manchester.

Elected

1851 †GASKOIN, GEORGE, Surgeon to the British Hospital for Diseases of the Skin; 7, Westbourne park. C. 1875-6. *Trans.* 2.

1819 GAULTER, HENRY.

1848 †GAY, JOHN, Senior Surgeon to the Great Northern Hospital, and Consulting Surgeon to the Asylum for Idiots; 34, Finsbury place. C. 1874-5.

1866 GEE, SAMUEL JONES, M.D., Physician to, and Lecturer on Medicine at, St. Bartholomew's Hospital; Physician to the Hospital for Sick Children; 54, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1871-6. *Trans.* 1.

1878 GERVIS, HENRY, M.D., Obstetric Physician to, and Lecturer on Obstetric Medicine at, St. Thomas's Hospital; Examiner in Obstetric Medicine at the University of London; 40, Harley street, Cavendish square.

1880 GIBBONS, ROBERT ALEXANDER, M.D., 32, Cadogan place.

1877 GODLEE, RICKMAN JOHN, Assistant-Surgeon to University College Hospital; and Demonstrator of Anatomy in University College; 81, Wimpole street, Cavendish square.

1870 GODSON, CLEMENT, M.D., Assistant-Physician-Accoucheur to St. Bartholomew's Hospital; 9, Grosvenor street, Grosvenor square.

1851 GOODFELLOW, STEPHEN JENNINGS, M.D., Consulting Physician to the Middlesex Hospital; Swinnerton Lodge, near Dartmouth, Devon. C. 1864-5. *Referee*, 1860-3. *Lib. Com.* 1863. *Trans.* 2.

1877 GOULD, ALFRED PEARCE, M.S., Assistant Surgeon to, and Lecturer on Anatomy at, the Westminster Hospital; Surgeon to the North-west London Hospital; 16, Queen Anne street, Cavendish square.

1873 GOWERS, WILLIAM RICHARD, M.D., Assistant Professor of Clinical Medicine in University College, and Assistant-Physician to University College Hospital; 50, Queen Anne street, Cavendish square. *Trans.* 6.

Elected

1851 GOWLLAND, PETER YEAMES, Surgeon to St. Mark's Hospital; Surgeon-Major Hon. Artillery Company; 34, Finsbury square.

1846 GREAM, GEORGE THOMPSON, M.D., Physician-Accoucheur to H.R.H. the Princess of Wales; Crawleydown park, Worth, Sussex. C. 1863.

1868 GREEN, T. HENRY, M.D., Physician to, and Lecturer on Pathology at, Charing Cross Hospital; Assistant-Physician to the Hospital for Consumption, Brompton; 74, Wimpole street, Cavendish square. *Referee*, 1882.

1875 GREENFIELD, WILLIAM SMITH, M.D., Professor of General Pathology in the University of Edinburgh; 7, Heriot row, Edinburgh. *Sci. Com.* 1879. *Referee*, 1881.

1843 †GREENHALGH, ROBERT, M.D., Consulting Physician to the Samaritan Free Hospital for Women and Children, and to the City of London Lying-in Hospital [27, Grosvenor street]. C. 1871-2. *Referee*, 1876-7. *Trans.* 1.

1860 GREENHOW, EDWARD HEADLAM, M.D., F.R.S., Consulting Physician to the Middlesex Hospital; and Consulting Physician to the Western General Dispensary; Castle Lodge, Reigate. C. 1876-7. *Referee*, 1870-5. *Trans.* 3.

1868 GRIGG, WILLIAM CHAPMAN, M.D., Assistant Obstetric Physician to the Westminster Hospital; Physician to the In-Patients, Queen Charlotte's Lying-in-Hospital; Assistant-Physician to the Victoria Hospital for Children; 6, Curzon street, Mayfair.

1852 GROVE, JOHN, Westgate court, Canterbury.

1860 GUENEAU DE MUSSY, HENRI, M.D.; 15, Rue du Cirque, Paris. *Lib. Com.* 1863-5.

1849 †GULL, SIR WILLIAM WITHEY, Bart., M.D., D.C.L., LL.D., F.R.S., Physician-Extraordinary to the Queen; Member of the Senate of the University of London; Consulting Physician to Guy's Hospital; 74, Brook street, Grosvenor square. C. 1864. V.P. 1874. *Referee*, 1855-63. *Trans.* 4.

1837 GULLY, JAMES MANBY, M.D.

Elected

1854 HABERSHON, SAMUEL OSBORNE, M.D., *Vice-President*, 70, Brook street, Grosvenor square. S. 1867. C. 1869-70. V.P. 1881. *Referee*, 1862-6, 1868, 1871-80. *Trans.* 3.

1881 HALL, FRANCIS DE HAVILLAND, M.D., Assistant Physician, and Physician to the Throat Department, Westminster Hospital; Physician to St. Mark's Hospital; 46, Queen Anne street, Cavendish square.

1870 HAMILTON, ROBERT, Surgeon to the South Hospital, Liverpool; 1 Prince's road, Liverpool.

1874 HARDIE, GORDON KENMURE, M.D., Deputy Inspector General of Hospitals; Florence road, Ealing, and Duff House, Banff, N.B.

1836 HARDING, JOHN FOSSE, Eagle House, Hornsey. C. 1858-9.

1856 HARE, CHARLES JOHN, M.D., late Professor of Clinical Medicine in University College, London, and late Physician to University College Hospital; Berkeley House, 15, Manchester square. C. 1873-4.

1857 HARLEY, GEORGE, M.D., F.R.S. 25, Harley street, Cavendish square. C. 1871-2. *Referee*, 1865-70, 1873-6. *Sci. Com.* 1862-3. *Trans.* 1.

1864 HARLEY, JOHN, M.D., F.L.S., Physician to, and Lecturer on General Anatomy and Physiology at, St. Thomas's Hospital; 39, Brook street, Grosvenor square. S. 1875-7. C. 1879-80. *Referee*, 1871-4, 1882. *Sci. Com.* 1879. *Trans.* 8.

1866 HARPER, PHILIP H., 30, Cambridge street, Hyde park.

1859 HARRIS, FRANCIS, M.D., F.L.S., 24, Cavendish square.

1880 HARRIS, VINCENT DORMER, M.D., Casualty Physician to St. Bartholomew's Hospital, and Assistant Physician to the Victoria Park Hospital; 39, Wimpole street, Cavendish square.

1872 HARRIS, WILLIAM H., M.D., Deputy Surgeon-General, Madras Army (retired); late Professor of Midwifery and Diseases of Women and Children, Madras Medical College; 78, Oxford gardens, Notting hill.

Elected

1870 HARRISON, REGINALD, Surgeon to the Liverpool Royal Infirmary, and Lecturer on Surgery at the School of Medicine; 38, Rodney street, Liverpool. *Trans.* 1.

1854 HAVILAND, ALFRED.

1870 HAWARD, J. WARRINGTON, Surgeon to St. George's Hospital; Surgeon to the Hospital for Sick Children; 16, Savile row, Burlington gardens. *Lib. Com.* 1881–2. *Trans.* 1.

1828 †HAWKINS, CÆSAR HENRY, F.R.S., Sergeant-Surgeon to H.M. the Queen, and Consulting Surgeon to St. George's Hospital; 26, Grosvenor street. C. 1830-1, 1860. V.P. 1838-9. T. 1841-4. P. 1855-6. *Referee*, 1843-7, 1850-4, 1857-9, 1861-82. *Trans.* 12.

1838 †HAWKINS, CHARLES, Inspector of Anatomical Schools in London; 27, Savile row, Burlington gardens. C. 1846-7. S. 1850. V.P. 1858. T. 1861-2. *Referee*, 1859-60. *Lib. Com.* 1843. *Trans.* 2.

1848 †HAWKSLEY, THOMAS, M.D., Consulting Physician to the Margaret street Dispensary for Consumption and Diseases of the Chest; 31, Grosvenor street.

1875 HAYES, THOMAS CRAWFORD, M.D., Assistant-Physician-Accoucheur and Assistant-Physician for Diseases of Women and Children to King's College Hospital; 17, Clarges street, Piccadilly.

1860 HAYWARD, HENRY HOWARD, Surgeon Dentist to, and Lecturer on Dental Surgery at, St. Mary's Hospital; 38, Harley street, Cavendish square. C. 1878-9.

1861 HAYWARD, WILLIAM HENRY, Chapel Ash, Wolverhampton.

1848 *HEALE, JAMES NEWTON, M.D.

1865 HEATH, CHRISTOPHER, Holme Professor of Clinical Surgery in University College, London; and Surgeon to University College Hospital; 36, Cavendish square. C. 1880. *Lib. Com.* 1870-3. *Trans.* 2.

1850 HEATON, GEORGE, M.D., Boston, U.S.

1882 HENSLEY, PHILIP J., M.D., Assistant Physician to St. Bartholomew's Hospital; 4, Henrietta street, Cavendish square.

Elected

1821 HERBERSKI, VINCENT, M.D., Professor of Medicine in the University of Wilna.

1877 HERMAN, GEORGE ERNEST, M.B., Assistant Obstetric Physician to the London Hospital; 7, West street, Finsbury circus.

1877 HERON, GEORGE ALLAN, M.D., Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; Assistant Physician to the West London Hospital for the Paralysed and Epileptic; 40, Margaret street, Cavendish square.

1843 HEWETT, PRESCOTT GARDNER, F.R.S., Serjeant-Surgeon-Extraordinary to H.M. the Queen; Surgeon in Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; Corresponding Member of the Academy of Medicine, and of the "Société de Chirurgie," Paris; 1, Chesterfield street, Mayfair. C. 1859. V.P. 1866-7. *Referee*, 1850-8, 1860-5, 1868-82. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 7.

1855 HEWITT, GRAILY, M.D., Professor of Midwifery in University College, London, and Obstetric Physician to University College Hospital; 36, Berkeley square. C. 1876. *Referee*, 1868-75, 1877-82. *Lib. Com.* 1868, 1874.

1880 HICKS, CHARLES CYRIL, M.D., 41, Cromwell Houses, South Kensington.

1873 HIGGENS, CHARLES, Assistant Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, Guy's Hospital; 38, Brook street, Grosvenor square. *Trans.* 2.

1862 HILL, M. BERKELEY, M.B. Lond., *Secretary*, Professor of Clinical Surgery in University College, London, and Surgeon to University College Hospital; Surgeon to the Lock Hospital; 55, Wimpole street, Cavendish square. C. 1878-9. S. 1881-2. *Trans.* 1.

1867 HILL, SAMUEL, M.D., 22, Mecklenburgh square.

1859 HIRD, FRANCIS, Consulting Surgeon to the Charing Cross Hospital; 13, Old Burlington street.

Elected

1861 *HOFFMEISTER, WILLIAM CARTER, M.D., Surgeon to H.M. the Queen in the Isle of Wight; Clifton House, Cowes, Isle of Wight.

1843 †HOLDEN, LUTHER, Consulting Surgeon to St. Bartholomew's Hospital; Consulting Surgeon to the Metropolitan Dispensary; Surgeon to the Foundling Hospital; Pinetoft, Ipswich. C. 1859. L. 1865. V.P. 1874. *Referee*, 1866-7. *Lib. Com.* 1858.

1879 HOLLAND, PHILIP ALEXANDER, M.A., Swancoe Park, Macclesfield.

1868 HOLLIS, WILLIAM AINSLIE, M.A., M.B., Camb., Assistant-Physician to the Sussex County Hospital; Park Gate, Preston road, Brighton.

1861 HOLMAN, WILLIAM HENRY, M.B. Lond., 68, Adelaide road, South Hampstead.

1856 HOLMES, TIMOTHY, M.A. Camb., *Vice-President*, Surgeon to St. George's Hospital; Surgeon-in-Chief to the Metropolitan Police Force; 18, Great Cumberland place, Hyde park. C. 1869-70. L. 1873-7. S. 1878-80. V.P. 1881-2. *Referee*, 1866-8, 1872. *Sci. Com.* 1867. *Lib. Com.* 1863-5. *Trans.* 8.

1846 †HOLT, BARNARD WIGHT, Consulting Surgeon to the Westminster Hospital; Medical Officer of Health for Westminster; 14, Savile row, Burlington gardens. C. 1862-3. V.P. 1879-80.

1846 †HOLTHOUSE, CARSTEN, 15, Percy street, Fitzroy square. C. 1863. *Referee* 1870-6. *Lib. Com.* 1859-60.

1878 HOOD, DONALD WILLIAM CHARLES, M.D. Cantab., Assistant Physician to the West London Hospital; 43, Green street, Park lane.

1878 HOUGHTON, WALTER B., M.D., Church Villa, Warrior square, St. Leonards-on-Sea.

1865 HOWARD, BENJAMIN, M.D., New York, U.S.

1865 HOWARD, EDWARD, M.D.

1881 HOWARD, HENRY, M.B., Stockwell Fever Hospital.

Elected

1874 HOWSE, HENRY GREENWAY, M.S. Lond., Surgeon to, and Lecturer on Anatomy at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; Examiner in Anatomy in the University of London; 10, St. Thomas's street, Southwark. *Sci. Com.* 1879. *Trans.* 2.

1877 *HUDSON, ROBERT SAMUEL, M.D., 58, West-end, Redruth, Cornwall.

1857 HULKE, JOHN WHITAKER, F.R.S., *Librarian*, Surgeon to the Middlesex Hospital; Surgeon to the Royal London Ophthalmic Hospital, Moorfields; 10, Old Burlington street. C. 1871-2. S. 1876-7. L. 1879-81. *Sci. Com.* 1867. *Lib. Com.* 1864-8. *Trans.* 8.

1857 HULME, EDWARD CHARLES, Woodbridge road, Guildford. *Trans.* 1.

1844 †HUMBY, EDWIN, M.D., 83, Hamilton terrace, St. John's wood. C. 1866-7.

1855 HUMPHRY, GEORGE MURRAY, M.D., F.R.S., Surgeon to Addenbrooke's Hospital; Professor of Human Anatomy and Physiology in the Cambridge University Medical School; Cambridge. *Trans.* 6.

1882 HUMPHRY, LAURENCE, M.B., City of London Hospital for Diseases of the Chest, Victoria park.

1873 HUNTER, WILLIAM GUYER, M.D., Hon. Surgeon to H.M. the Queen; late Principal of, and Professor of Medicine in, Grant Medical College, Bombay; Surgeon-General Bombay Army; 21, Norfolk crescent, Hyde park.

1849 HUSSEY, EDWARD LAW, Senior Surgeon to the Radcliffe Infirmary, and Consulting Surgeon to the County Lunatic Asylum and the Warneford Asylum; 8, St. Aldate's, Oxford. *Trans.* 1.

1856 HUTCHINSON, JONATHAN, F.R.S., *Vice-President*, Senior Surgeon to the London Hospital; Consulting Surgeon to the Royal London Ophthalmic Hospital, Moorfields; and Surgeon to the Hospital for Diseases of the Skin; 15, Cavendish square. C. 1870. V.P. 1882. *Referee*, 1876-81. *Lib. Com.* 1864-5. *Trans.* 10: *Pro.* 2.

Elected

1820 HUTCHINSON, WILLIAM, M.D.

1840 †HUTTON, CHARLES, M.D., Consulting Physician to the General Lying-in Hospital; 26, Lowndes street, Belgrave square. C. 1858-9.

1866 ILES, FRANCIS HENRY WILSON, M.D., Watford, Herts.

1847 IMAGE, WILLIAM EDMUND, Consulting Surgeon to the Suffolk General Hospital; Bury St. Edmund's, Suffolk. *Trans.* 1.

1856 INGLIS, CORNELIUS, M.D., Cairo. [Athenæum Club, Pall Mall.]

1871 JACKSON, J. HUGHLINGS, M.D., F.R.S., Physician to the London Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 3, Manchester square.

1841 †JACKSON, PAUL, 51, Wellington road, St. John's Wood. C. 1862.

1863 JACKSON, THOMAS VINCENT, Surgeon to the South Staffordshire General Hospital; Darlington st., Wolverhampton.

1841 JACOBOVICS, MAXIMILIAN MORITZ, M.D., Vienna.

1825 JAMES, JOHN B., M.D.

1851 †JENNER, SIR WILLIAM, Bart., M.D., K.C.B., D.C.L., LL.D., F.R.S., Physician in Ordinary to H.M. the Queen, and to H.R.H. the Prince of Wales; Emeritus Professor of Clinical Medicine in University College, London; and Consulting Physician to University College Hospital; Member of the Senate of the University of London; 63, Brook street, Grosvenor square. C. 1864. V.P. 1875. *Referee*, 1855, 1859-63. *Trans.* 3.

1881 JENNINGS, WILLIAM OSCAR, M.D., 8, Rue Roy, Paris.

1851 JOHNSON, EDMUND CHARLES, Corresponding Member of the Medical and Philosophical Society of Florence, and of "l'Institut Génevois."

1847 †JOHNSON, GEORGE, M.D., F.R.S., Physician to King's College Hospital; Member of the Senate of the University of London; 11, Savile row, Burlington gardens. C. 1862-3. V.P. 1870. L. 1878-80. *Referee*, 1853-61, 1864-9. *Lib. Com.* 1860-1. *Trans.* 10.

Elected

1881 JOHNSON, GEORGE LINDSAY, M.A., Fern Lea, Highfield hill, Upper Norwood.

1868 JOHNSTON, WILLIAM, M.D., 21, Upper Grosvenor road, Tunbridge Wells.

1848 JOHNSTONE, ATHOL ARCHIBALD WOOD, Consulting Surgeon to the Brighton Hospital for Sick Children, St. Moritz House, 61, Dyke road, Brighton. *Lib. Com.* 1860. *Trans.* 1.

1876 JONES, LESLIE, M.D., Medical Officer of Health for Blackpool; 3, Brighton Parade, Blackpool, Lancashire.

1875 *JONES, PHILIP SYDNEY, M.D., Consulting Surgeon to the Sydney Infirmary; Examiner in Medicine, Sydney University; 10, College street, Sydney, New South Wales. [Agents: Messrs. D. Jones & Co., 1, Gresham buildings, Basinghall street.]

1837 †JONES, THOMAS WILLIAM, M.D., Bylocks, Enfield Highway. C. 1858.

1859 JONES, WILLIAM PRICE, M.D., Claremont road, Surbiton, Kingston.

1865 JORDAN, FURNEAUX, Surgeon to the Queen's Hospital, and Professor of Surgery at the Queen's College, Birmingham; 22, Colmore row, Birmingham.

1881 JULER, HENRY EDWARD, Assistant Surgeon Royal Westminster Ophthalmic Hospital; 77, Wimpole street, Cavendish square.

1816 *KAUFFMANN, GEORGE HERMANN, M.D., Hanover.

1882 KEETLEY, CHARLES R. B., Assistant Surgeon to the West London Hospital; 20, Princes street, Hanover square.

1872 KELLY, CHARLES, M.D., Professor of Hygiene at King's College, London, and Medical Officer of Health for the West Sussex Combined Sanitary District, Worthing, Sussex.

1848 *KENDELL, DANIEL BURTON, M.D., Heath House, Wakefield, Yorkshire.

Elected

1877 *KHORY, RUSTONJEE NASERWANJEE, M.D. Brussels; Physician to the Parell Dispensary, Bombay; Lecturer to Native Midwives, Grant Medical College, Bombay; 39, St. James's square, Holland park.

1857 KIALLMARK, HENRY WALTER, 5, Pembridge gardens, Bayswater.

1881 KIDD, PERCY, M.B., Casualty Physician to St. Bartholomew's Hospital, and Assistant Physician to the Hospital for Consumption, Brompton; 60, Brook street, Grosvenor square. *Trans.* 1.

1882 KING, DAVID ALEXANDER, 57, Pembridge villas, Bayswater.

1851 †KINGDON, JOHN ABERNETHY, Surgeon to the City of London Truss Society, and to the City Dispensary; 2, New Bank buildings, Lothbury. C. 1866-7. V.P. 1872-3. *Sci. Com.* 1867. *Trans.* 1.

1855 LANE, JAMES ROBERT, Consulting Surgeon to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1870. *Referee*, 1869, 1877-82. *Lib. Com.* 1869. *Trans.* 1.

1840 †LANE, SAMUEL ARMSTRONG, Consulting Surgeon to St. Mary's Hospital and to the Lock Hospital; 49, Norfolk square, Hyde park. C. 1849-50. V.P. 1865. *Referee*, 1850.

1882 LANG, WILLIAM, Ophthalmic Surgeon to the Middlesex Hospital; 26, Upper Wimpole street, Cavendish square.

1865 LANGTON, JOHN, Surgeon to, and Lecturer on Anatomy at, St. Bartholomew's Hospital; Surgeon to the City of London Truss Society; 2, Harley street, Cavendish square. C. 1881. *Lib. Com.* 1879-80.

1873 *LARCHER, O., M.D., Laureate of the Institute of France, of the Medical Faculty, and Academy of Paris, &c.; 97, Rue de Passy, Passy, Paris.

1841 *LASHMAR, CHARLES, M.D., 83, North End, Croydon, Surrey.

Elected

1862 LATHAM, PETER WALLWORK, M.A., M.D., Downing Professor of Medicine, Cambridge University; Physician to Addenbrooke's Hospital, Cambridge; 17, Trumpington street, Cambridge.

1816 LAWRENCE, G. E.

1880 LAYCOCK, GEORGE LOCKWOOD, M.B., 12, Upper Berkeley street, Portman square.

1843 †LEE, HENRY, Consulting Surgeon to St. George's Hospital; 9, Savile row, Burlington gardens. C. 1856-7. L. 1863-4. V.P. 1868-9. *Referee*, 1855, 1866-8. *Sci. Com.* 1867. *Trans.* 13. *Pro.* 2.

1877 LEESON, ARTHUR EDMUND, M.A., M.D. [South America.]

1869 LEGG, JOHN WICKHAM, M.D., Assistant Physician to, and Lecturer on Pathological Anatomy at, St. Bartholomew's Hospital; 47, Green street, Park lane. *Referee*, 1882. *Lib. Com.* 1878-82. *Trans.* 2.

1836 LEIGHTON, FREDERICK, M.D.

1872 LIEBREICH, RICHARD, Consulting Ophthalmic Surgeon to St. Thomas's Hospital; Paris.

1806 LIND, JOHN, M.D.

1878 LISTER, JOSEPH, D.C.L., LL.D., F.R.S., Surgeon Extraordinary to H.M. the Queen; Professor of Clinical Surgery at King's College, London; and Surgeon to King's College Hospital; 12, Park crescent, Regent's park.

1872 *LITTLE, DAVID, M.D., Surgeon to the Royal Eye Hospital, Manchester; 21, St. John's street, Manchester.

1871 LITTLE, LOUIS STROMEYER, Shanghai, China.

1870 LIVINGSTON, JOHN, M.D., New Barnet, Hertfordshire.

1819 LLOYD, ROBERT, M.D.

1820 LOCHER, J. G., M.C.D., Town Physician of Zurich. *Trans.* 2.

1881 LOCKWOOD, CHARLES BARRETT, Demonstrator of Anatomy at St. Bartholomew's Hospital: 8, Serjeants' inn, Fleet street.

Elected

1860 LONGMORE, THOMAS, C.B., Hon. Surgeon to H.M. the Queen; Surgeon-General, Army Medical Staff, and Professor of Military Surgery, Army Medical School, Netley, Southampton; Woolston Lawn, Woolston, Hants. *Trans.* 2.

1836 LÖWENFELD, JOSEPH S., M.D., Berbice.

1871 LOWNDS, THOMAS MACKFORD, M.D., late Professor of Anatomy and Physiology at Grant Medical College, Bombay; Egham Hill, Surrey.

1877 LOWNE, BENJAMIN THOMPSON, Lecturer on Physiology, Middlesex Hospital Medical School; 65, Cambridge gardens, Notting hill.

1881 LUCAS, RICHARD CLEMENT, Senior Assistant Surgeon to, and Demonstrator of Operative and Practical Surgery at, Guy's Hospital; Surgeon to the Evelina Hospital for Sick Children; 18, Finsbury square.

1857 LYON, FELIX WILLIAM, M.D., 118, Princes street, Edinburgh.

1882 LYONS, ISIDORE, Dental Surgeon to the Evelina Hospital for Children; Assistant Dental Surgeon to St. Bartholomew's Hospital; 19, Queen Anne street, Cavendish square.

1867 MABERLY, GEORGE FREDERICK, 98, Collins street east, Melbourne, Victoria.

1873 MACCARTHY, JEREMIAH, M.A., Surgeon to, and Lecturer on Physiology at, the London Hospital; Examiner in Surgery in the University of London; 15, Finsbury square. *Lib. Com.* 1882.

1867 MACCORMAC, SIR WILLIAM, M.A., Surgeon to, and Lecturer on Surgery at, St. Thomas's Hospital; Examiner in Surgery at the University of London; 13, Harley street. *Trans.* 1.

1862 *M'DONNELL, ROBERT, M.D., F.R.S., Surgeon to Steevens' Hospital; 89, Merrion square west, Dublin. *Trans.* 2.

Elected

1880 *MACFARLANE, ALEXANDER WILLIAM, M.D., Consulting Physician to the Kilmarnock Fever Hospital and Infirmary; Walmer, Kilmarnock, N.B.

1866 MACGOWAN, ALEXANDER THORBURN, Vyvyan House, Clifton, near Bristol.

1880 MCHARDY, MALCOLM MACDONALD, Ophthalmic Surgeon to King's College Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 5, Savile row.

1822 MACINTOSH, RICHARD, M.D.

1859 *M'INTYRE, JOHN, M.D., Odiham, Hants.

1873 MACKELLAR, ALEXANDER OBERLIN, M.S.I., Assistant Surgeon to St. Thomas's Hospital; 22, George street, Hanover square.

1881 MACKENZIE, STEPHEN, M.D., Senior Assistant Physician, and Physician in charge of Department of Skin Diseases at the London Hospital; 26, Finsbury sqnare.

1876 MACKEY, EDWARD, M.D., 123, Western road, Brighton.

1854 *MACKINDER, DRAPER, M.D., Consulting Surgeon to the Dispensary, Gainsborough, Lincolnshire.

1879 MACLAGAN, THOMAS JOHN, M.D., Physician-in-Ordinary to their R.H. the Prince and Princess Christian of Schleswig-Holstein; 9, Cadogan place, Belgrave square.

1860 MACLEAN, JOHN, M.D., 24, Portman street, Portman square.

1876 MACNAMARA, CHARLES, Surgeon to, and Lecturer on Surgery at, the Westminster Hospital; Surgeon-Major Bengal Medical Service; Fellow of the Calcutta University; 13, Grosvenor street.

1842 MACNAUGHT, JOHN, M.D., 74, Huskisson street, Liverpool.

1881 MACREADY, JONATHAN F. C. H., Surgical Registrar to St. Bartholomew's Hospital; 125, Harley street, Cavendish square.

1880 MADDICK, EDMUND D., Royal Naval Hospital, Haslar, Gosport; 184, Brixton road.

Elected

1880 MAKINS, GEORGE HENRY, Blackheath park.

1876 MALLAM, BENJAMIN, Meadow Side, Leacroft road, Staines.

1855 MARCET, WILLIAM, M.D., F.R.S.; 39, Grosvenor street. C. 1871. *Referee*, 1866-70. *Sci. Com.* 1863. *Lib. Com.* 1866-8. *Trans.* 3.

1867 MARSH, F. HOWARD, Assistant Surgeon to St. Bartholomew's Hospital; Assistant Surgeon to the Hospital for Sick Children, Great Ormond street; 36, Bruton street, Berkeley square. C. 1882. *Lib. Com.* 1880-81. *Trans.* 2.

1838 MARSH, THOMAS PARR, M.D.

1851 MARSHALL, JOHN, F.R.S., *President*, Professor of Anatomy to the Royal Academy of Arts; Professor of Surgery in University College, London, and Surgeon to University College Hospital; 10, Savile row, Burlington gardens. C. 1866. V.P. 1875-6. P. 1881. *Referee*, 1867, 1871-4, 1877-81. *Trans.* 2.

1864 MASON, FRANCIS, Surgeon to, and Lecturer on Operative Surgery at, St. Thomas's Hospital; 5, Brook street, Grosvenor square. C. 1880-81. *Trans.* 1.

1839 MEADE, RICHARD HENRY, Consulting Surgeon to the Bradford Infirmary; Bradford, Yorkshire. *Trans.* 1.

1870 MEADOWS, ALFRED, M.D., Physician-Accoucheur to, and Lecturer on Midwifery at, St. Mary's Hospital; 27, George street, Hanover square. *Lib. Com.* 1875-7.

1865 MEDWIN, AARON GEORGE, M.D., Dental Surgeon to the Royal Kent Dispensary, 34, Bruton street, Berkeley square, and 11, Montpellier row, Blackheath, Kent.

1880 MEREDITH, WILLIAM APPLETON, M.B., C.M., Assistant Surgeon to the Samaritan Free Hospital for Women and Children; 6, Queen Anne street, Cavendish square.

1867 MEREDYTH, COLOMIATI, M.D., 10, George street, Hanover square.

1874 MERRIMAN, JOHN J., 45, Kensington square.

Elected

1815 MEYER, AUGUSTUS, M.D., St. Petersburg.

1840 MIDDLEMORE, RICHARD, Consulting Surgeon to the Birmingham Eye Hospital; 19, Temple row, Birmingham.

1854 MIDDLESHIP, EDWARD ARCHIBALD.

1882 MILLS, JOSEPH, 15, Henrietta street, Cavendish square.

1873 MILNER, EDWARD, Surgeon for Out-Patients to the Lock Hospital ; 32, New Cavendish street, Portland place.

1844 †MONTEFIORE, NATHANIEL, 18, Portman square.

1873 MOORE, NORMAN, M.D., Warden of the College and Lecturer on Comparative Anatomy, Demonstrator of Morbid Anatomy, St. Bartholomew's Hospital; the College, St. Bartholomew's Hospital.

1857 MORGAN, JOHN, 3, Sussex place, Hyde park gardens. C. 1880-1. *Lib. Com.* 1862-3. *Trans.* 1.

1861 MORGAN, JOHN EDWARD, M.D., Physician to the Manchester Royal Infirmary, and Professor of Medicine in the Owens College, Manchester; 1, St. Peter's square, Manchester.

1878 MORGAN, JOHN HAMMOND, M.A., Assistant Surgeon to the Charing Cross Hospital, and to the Hospital for Sick Children, Great Ormond street; 12, Chapel street, Park lane. *Trans.* 1.

1874 MORRIS, HENRY, M.A. Lond., Surgeon to, and Lecturer on Surgery at, the Middlesex Hospital; 2, Mansfield street, Portland place. *Referee*, 1882. *Trans.* 6.

1879 MORRIS, MALCOLM ALEXANDER, Lecturer on Skin Diseases at St. Mary's Hospital; 63, Montagu square.

1851 MOUAT, FREDERIC JOHN, M.D., Deputy Inspector-General of Hospitals; Medical Inspector to the Local Government Board; and Member of the Senate of the University of Calcutta; 12, Durham villas, Kensington.

1868 MOXON, WALTER, M.D., F.L.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 6, Finsbury Circus. *Referee*, 1879-82. *Trans.* 1.

Elected

1879 MUNK, WILLIAM, M.D., Harveian Librarian, Royal College of Physicians; Consulting Physician to the Roya. Hospital for Incurables; 40, Finsbury square.

1875 MURPHY, WILLIAM KIRKPATRICK, M.A., M.D., 29, Queen Anne street, Cavendish square.

1873 MURRAY, IVOR, M.D., F.R.S. Ed., 8, Huntriss Row, Scarborough.

1880 MURRELL, WILLIAM, M.D., Assistant Physician to the Royal Hospital for Diseases of the Chest; Lecturer on Materia Medica and Therapeutics at the Westminster Hospital; 38, Weymouth street, Portland place. *Trans.* 1.

1863 MYERS, ARTHUR BOWEN RICHARDS, Surgeon to the 1st Battalion, Coldstream Guards; Hospital, Vincent square, Westminster. C. 1878-9. *Lib. Com.* 1877.

1882 MYERS, A. T., M.D., Medical Registrar, St. George's Hospital; 12, Hereford gardens, Oxford street.

1831 NALL, SAMUEL, M.B., Casualty Physician to St. Bartholomew's Hospital; 34, Highgate road.

1870 NEILD, JAMES EDWARD, M.D., Lecturer on Forensic Medicine in the University of Melbourne; 166, Collins street east, Melbourne, Victoria.

1835 †NELSON, THOMAS ANDREW, M.D., 10, Nottingham terrace, York gate, Regent's park. *Lib. Com.* 1841.

1877 NETTLESHIP, EDWARD, Ophthalmic Surgeon to, and Lecturer on Ophthalmology at, St. Thomas's Hospital; Ophthalmic Surgeon to the Hospital for Sick Children; 5, Wimpole street, Cavendish square.

1843 †NEWTON, EDWARD, 85, Gloucester terrace, Hyde Park. C. 1863-4.

1868 NICHOLLS, JAMES, M.D., Duke street, Chelmsford, Essex.

1849 NORMAN, HENRY BURFORD, Portland Lodge, Southsea, Hants. *Lib. Com.* 1857.

1847 *NOURSE, WILLIAM EDWARD CHARLES, late Surgeon to the Brighton Children's Hospital; Bouverie House, Mount Radford, Exeter.

Elected

1864 NUNN, THOMAS WILLIAM, Consulting Surgeon to the Middlesex Hospital; 8, Stratford place, Oxford street.

1870 NUNNELEY, FREDERICK BARHAM, M.D. *Trans.* 2.

1880 O'CONNOR, BERNARD, A.B., M.D., 40, Brook street, Grosvenor square.

1847 O'CONNOR, THOMAS, March, Cambridgeshire.

1880 OGILVIE, GEORGE, M.B., Lecturer on Experimental Physics at the Westminster Hospital; 27, Welbeck street, Cavendish square.

1880 OGILVIE, LESLIE, M.B., Lecturer on Comparative Anatomy at the Westminster Hospital; 46, Welbeck street, Cavendish square.

1858 OGLE, JOHN WILLIAM, M.D., Consulting Physician to St. George's Hospital; 30, Cavendish square. C. 1873. *Referee*, 1864-72. *Trans.* 4.

1855 *OGLE, WILLIAM, M.A., M.D., Physician to the Derby Infirmary; The Elms, Duffield road, Derby.

1860 OGLE, WILLIAM, M.D., Superintendent of Statistics in the Registrar-General's Department, Somerset House; 10, Gordon street, Gordon square. S. 1868-70. C. 1876-7. *Lib. Com.* 1871-5. *Trans.* 4.

1870 OLDHAM, CHARLES FREDERIC, India [Agents: Messrs. Grindlay and Co., 55, Parliament street].

1871 *O'NEILL, WILLIAM, M.D., Physician to the Lincoln Lunatic Hospital, Lincoln.

1873 ORD, WILLIAM MILLER, M.D., Physician to, and Lecturer on Medicine at, St. Thomas's Hospital; 7, Brook street, Hanover square. *Trans.* 6.

1877 ORMEROD, JOSEPH ARDERNE, M.B., Casualty Physician to St. Bartholomew's Hospital; 25, Upper Wimpole street.

1875 OSBORN, SAMUEL, 10, Maddox street, Regent street, and 17, Gresham park, Brixton.

1879 OWEN, EDMUND, Surgeon to St. Mary's Hospital; Surgeon to the Hospital for Sick Children; 49, Seymour street, Portman square. *Trans.* 1.

Elected

1882 OWEN, HERBERT ISAMBARD, M.D., Assistant Physician to the Hospital for Consumption, Brompton; Lecturer on Materia Medica at St. George's Hospital; 41, Gloucester gardens.

1874 PAGE, HERBERT WILLIAM, M.A., M.C. Cantab., Surgeon (with charge of out-patients) to, and Lecturer on Operative and Practical Surgery at, St. Mary's Hospital; 146, Harley street, Cavendish square. *Trans.* 1.

1847 *PAGE, WILLIAM BOUSFIELD, Consulting Surgeon to the Cumberland Infirmary, Carlisle. *Trans.* 2.

1840 †PAGET, SIR JAMES, Bart., D.C.L., LL.D., F.R.S., Sergeant-Surgeon to H.M. the Queen; Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. Bartholomew's Hospital; Member of the Senate of the University of London; 1, Harewood place, Hanover square. C. 1848-9. V.P. 1861. T. 1867. P. 1875-6. *Referee*, 1844-6, 1848, 1851-60, 1862-6, 1868-74. *Sci. Com.* 1863. *Lib. Com.* 1846-7. *Trans.* 12.

1858 *PALEY, WILLIAM, M.D., Physician to the Ripon Dispensary; Ripon, Yorkshire.

1847 PARKER, NICHOLAS, M.D., Paris.

1873 PARKER, ROBERT WILLIAM, Assistant-Surgeon to the East London Children's Hospital; 8, Old Cavendish-street. *Trans.* 2.

1841 PARKIN, JOHN, M.D., 5, Codrington place, Brighton.

1865 PAVY, FREDERICK WILLIAM, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; 35, Grosvenor street. *Referee*, 1871-82. *Trans.* 1.

1869 PAYNE, JOSEPH FRANK, M.D., Assistant-Physician to, and Lecturer on Pathological Anatomy at, St. Thomas's Hospital; 78, Wimpole street, Cavendish square. *Sci. Com.* 1879. *Lib. Com.* 1878-82.

1879 PEEL, ROBERT, L.K.Q.C.P.I., 120, Collins street east, Melbourne, Victoria.

1856 PEIRCE, RICHARD KING, Woodside, Windsor forest, Berks.

1830 PELECHIN, CHARLES P., M.D., St. Petersburg.

Elected

1855 *PEMBERTON, OLIVER, Surgeon to the Birmingham General Hospital, and Professor of Surgery at the Queen's College, Birmingham; 12, Temple row, Birmingham. *Trans.* 1.

1874 PENHALL, JOHN THOMAS, 5, Eversfield place, St. Leonard's, Sussex.

1870 PERRIN, JOHN BESWICK, late Medical Tutor and Demonstrator of Practical and Surgical Anatomy, Owen's College; Vernon House, Leigh, Lancashire.

1879 *PESIKAKA, HORMASJI DOSABHAI, L.R.C.P. Glasg., Marine Lines, Bombay.

1878 *PHILIPSON, GEORGE HARE, M.D., M.A. Cantab., D.C.L. Durh., Professor of Medicine at Durham University; Senior Physician to the Newcastle-upon-Tyne Infirmary; 7, Eldon square, Newcastle-upon-Tyne.

1852 PHILLIPS, RICHARD, 27, Leinster square, Bayswater. C. 1877.

1867 PICK, THOMAS PICKERING, Surgeon to, and Lecturer on Surgery at, St. George's Hospital; 13, South Eaton place, Eaton square. *Referee*, 1882. *Sci. Com.* 1870. *Lib. Com.* 1879-81.

1841 †PITMAN, HENRY ALFRED, M.D., Consulting Physician to St. George's Hospital, and to the Royal General Dispensary, St. Pancras; 28, Gordon square. L. 1851-3. C. 1861-2. T. 1863-8. V.P. 1870-1. *Referee*, 1849-50. *Lib. Com.* 1847.

1871 POLLOCK, ARTHUR JULIUS, M.D., Physician to, and Lecturer on the Principles and Practice of Medicine at, Charing Cross Hospital; Physician to the Foundling Hospital; 85, Harley street, Cavendish square.

1845 †POLLOCK, GEORGE DAVID, Surgeon-in-Ordinary to H.R.H. the Prince of Wales; Consulting Surgeon to St. George's Hospital; 36, Grosvenor street. C. 1856-7. L.-1859-62. V.P. 1870-1. *Referee*, 1858, 1864-9, 1877-82. *Trans.* 4.

1865 POLLOCK, JAMES EDWARD, M.D., Physician to the Hospital for Consumption, Brompton; 52, Upper Brook street, Grosvenor square. C. 1882. *Referee*, 1872-81.

Elected

1871 POORE, GEORGE VIVIAN, M.D., Professor of Medical Jurisprudence in University College, London; Assistant-Physician to University College Hospital; Physician to the Royal Infirmary for Children and Women, Waterloo road; Examiner in Forensic Medicine in the University of London; 30, Wimpole street. *Trans.* 1.

1846 POTTER, JEPHSON, M.D., F.L.S.

1842 POWELL, JAMES, M.D.

1867 POWELL, RICHARD DOUGLAS, M.D., Physician to the Middlesex Hospital; Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 62, Wimpole street, Cavendish sq. *Referee*, 1879-82. *Trans.* 2.

1867 POWER, HENRY, Ophthalmic Surgeon to, and Lecturer on Ophthalmic Surgery at, St. Bartholomew's Hospital; 37A, Gt. Cumberland place, Hyde park. C. 1882. *Referee*, 1870-81. *Sci. Com.* 1870. *Lib. Com.* 1872-8.

1857 PRIESTLEY, WILLIAM OVEREND, M.D., Consulting Physician-Accoucheur to King's College Hospital, and to the St. Marylebone Infirmary; 17, Hertford street, Mayfair. C. 1874-5. *Referee*, 1867-73, 1877-82. *Sci. Com.* 1863.

1874 PURVES, WILLIAM LAIDLAW, Aural Surgeon to Guy's Hospital; 20, Stratford place, Oxford street. *Trans.* 2.

1878 PYE, WALTER, Surgeon (with charge of out-patients) to St. Mary's Hospital; 4, Sackville street, Piccadilly.

1877 PYE-SMITH, PHILIP HENRY, M.D., Assistant-Physician to, and Lecturer on Physiology at, Guy's Hospital; Examiner in Physiology at the University of London; 54, Harley street, Cavendish square.

1850 †QUAIN, RICHARD, M.D., F.R.S., Consulting Physician to the Hospital for Consumption, Brompton; Member of the Senate of the University of London; 67, Harley street, Cavendish square. C. 1866-7. V.P. 1878-9. *Sci. Com.* 1863. *Trans.* 1.

Elected

1835 †QUAIN, RICHARD, F.R.S., Surgeon-Extraordinary to H.M. the Queen; Emeritus Professor of Clinical Surgery, University College, London, and Consulting Surgeon to University College Hospital; 32, Cavendish square. C. 1838-9. L. 1846-8. T. 1851-3. V.P. 1856-7. *Referee*, 1845-6, 1848, 1858-9. *Lib. Com.* 1846. *Trans.* 1. *Pro.* 2.

1852 RADCLIFFE, CHARLES BLAND, M.D., *Treasurer*, Consulting Physician to the Westminster Hospital; Physician to the National Hospital for the Paralysed and Epileptic; 25, Cavendish square. C. 1867-8. V.P. 1879-80. T. 1881-2. *Referee*, 1862-6, 1870-8.

1871 RALFE, CHARLES HENRY, M.D., M.A., Assistant Physician to the London Hospital, and late Physician to the Seamen's Hospital, Greenwich; 26, Queen Anne street, Cavendish square.

1857 RANKE, HENRY, M.D., 3, Sophienstrasse, Munich.

1854 RANSOM, WILLIAM HENRY, M.D., F.R.S., Physician to the Nottingham General Hospital, Nottingham.

1869 READ, THOMAS LAURENCE, 57, Gloucester road [11, Petersham terrace], Queen's gate, South Kensington.

1858 REED, FREDERICK GEORGE, M.D., 46, Hertford street, Mayfair. *Trans.* 1.

1821 REEDER, HENRY, M.D., Varick, Seneca County, New York, United States.

1857 REES, GEORGE OWEN, M.D., F.R.S., Consulting Physician to Guy's Hospital; 26, Albemarle street, Piccadilly. C. 1873. *Referee*, 1860-72, 1875-81. *Trans.* 1.

1882 REID, JAMES, M.D., Resident Physician to H.M. the Queen, Windsor Castle.

1855 REYNOLDS, JOHN RUSSELL, M.D., F.R.S., Physician-in-Ordinary to H.M.'s Household; Consulting-Physician to University College Hospital; 38, Grosvenor street. C. 1870. *Referee*, 1867-9.

1865 RHODES, GEORGE WINTER, Surgeon to the Huddersfield Infirmary; Queen street south, Huddersfield.

Elected

1881 RICE, GEORGE, M.B., C.M. Edinb., The Infirmary, Plumstead, Kent.

1852 RICHARDSON, CHRISTOPHER THOMAS, M.B., 13, Nelson crescent, Ramsgate.

1845 †RIDGE, BENJAMIN, M.D., 8, Mount street, Grosvenor square.

1863 RINGER, SYDNEY, M.D., Professor of the Principles and Practice of Medicine in University College, London, and Physician to University College Hospital; 15, Cavendish place, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans.* 5.

1871 RIVINGTON, WALTER, M.S., Surgeon to, and Lecturer on Anatomy at, the London Hospital; 22, Finsbury square. *Trans.* 2.

1871 *ROBERTS, DAVID LLOYD, M.D., Physician to St. Mary's Hospital, Manchester; 23, St. John's street, Deansgate, Manchester.

1878 ROBERTS, FREDERICK THOMAS, M.D., Professor of Materia Medica and Therapeutics in University College, London and Physician to University College Hospital; Examiner in Materia Medica in the University of London; 53, Harley street, Cavendish square, W.

1857 ROBERTSON, JOHN CHARLES GEORGE, Medical Superintendent of the Cavan District Lunatic Asylum; Monaghan, Ireland.

1873 ROBERTSON, WILLIAM H., M.D., Consulting Physician to the Devonshire Hospital and Buxton Bath Charity; Buxton, Derbyshire.

1843 RODEN, WILLIAM, M.D., Morningside, Kidderminster, Worcestershire.

1850 ROPER, GEORGE, M.D., Consulting Physician to the Eastern Division of the Royal Maternity Charity; Physician to the Royal Infirmary for Children and Women, Waterloo Bridge road; 19, Ovington gardens, S.W. C. 1879-80.

1857 ROSE, HENRY COOPER, M.D., F.L.S., Surgeon to the Hampstead Dispensary; Penrose House, Hampstead. *Trans.* 1.

Elected

1882 ROUTH, AMAND J. McC., M.B., B.S., 6, Upper Montagu street, W.

1849 †ROUTH, CHARLES HENRY FELIX, M.D., Physician to the Samaritan Free Hospital for Women and Children; 52, Montagu square. *Lib. Com.* 1854-5. *Trans.* 1.

1863 ROWE, THOMAS SMITH, M.D., Surgeon to the Royal Sea-Bathing Infirmary; Cecil street, Margate, Kent.

1882 ROY, CHARLES L., M.D., The Brown Institution, Wandsworth road.

1845 RUSSELL, JAMES, M.D., Physician to the Birmingham General Hospital; 91, New Hall street, Birmingham.

1871 RUTHERFORD, WILLIAM, M.D., F.R.S., Professor of Physiology in the University of Edinburgh; 14, Douglas crescent, Edinburgh.

1856 SALTER, S. JAMES A., M.B., F.R.S., F.L.S., late Lecturer on Dental Surgery at Guy's Hospital; Basingfield, near Basingstoke, Hants. C. 1871. *Lib. Com.* 1878. *Trans.* 2.

1849 †SANDERSON, HUGH JAMES, M.D., 26, Upper Berkeley street, Portman square. C. 1872-3. *Lib. Com.* 1862-3.

1855 SANDERSON, JOHN BURDON, M.D., LL.D., F.R.S., *Vice-President*, Jodrell Professor of Human Physiology and Histology at University College, London; 26, Gordon square. C. 1869-70. V.P. 1882. *Referee*, 1867-8, 1876-81. *Sci. Com.* 1862, 1870. *Lib. Com.* 1876-81. *Trans.* 2.

1867 SANDFORD, FOLLIOTT JAMES, M.D., Market Drayton, Shropshire.

1879 SANGSTER, ALFRED, B.A., M.B., Lecturer on Skin Diseases at the Charing Cross Hospital; 7, Old Burlington street. *Trans.* 1.

1847 †SANKEY, WILLIAM HENRY OCTAVIUS, M.D., Boreatton park, Shrewsbury.

1869 SANSOM, ARTHUR ERNEST, M.D., Physician (with charge of out-patients) to the London Hospital; 84, Harley street, Cavendish square. *Trans.* 2.

Elected

1845 †SAUNDERS, EDWIN, Surgeon-Dentist to H.M. the Queen, and to H.R.H. the Prince of Wales; 13A, George street, Hanover square. C. 1872-3.

1834 SAUVAN, LUDWIG V., M.D., Warsaw.

1879 SAVAGE, GEORGE HENRY, M.D., Bethlem Royal Hospital, St. George's road, Southwark.

1859 SAVORY, WILLIAM SCOVELL, F.R.S., Surgeon to, and Lecturer on Surgery at, St. Bartholomew's Hospital; Surgeon to Christ's Hospital; 66, Brook street, Grosvenor square. C. 1871-2. L. 1878. *Referee*, 1865-70, 1873-77, 1879-82. *Sci. Com.* 1862, 1867, 1870. *Lib. Com.* 1866-8. *Trans.* 6.

1873 SCOTT, JOHN MOORE JOHNSTON, M.D., Lurgan, County Armagh.

1861 *SCOTT, WILLIAM, M.D., Physician to the Huddersfield Infirmary; Waverley House, Huddersfield.

1882 SCRIVEN, JOHN BARCLAY, Brigade Surgeon, Bengal (retired), late Professor of Anatomy, Surgery, and Ophthalmic Surgery at the Lahore Medical School; 95, Oxford gardens, Notting hill.

1863 SEDGWICK, WILLIAM, 12, Park place, Upper Baker street. *Trans.* 2.

1877 SEMON, FELIX, M.D., Assistant Physician for Diseases of the Throat to St. Thomas's Hospital; 59, Welbeck street, Cavendish square. *Trans.* 1.

1875 SEMPLE, ROBERT HUNTER, M.D., Physician to the Bloomsbury Dispensary; 8, Torrington square. *Sci. Com.* 1879.

1873 *SHAPTER, LEWIS, B.A., M.B., Physician to the Devon and Exeter Hospital; the Barnfield, Exeter.

1882 SHARKEY, SEYMOUR J., M.B., Assistant Physician to St. Thomas's Hospital; 77, Lambeth Palace road.

1840 SHARP, WILLIAM, M.D., F.R.S., Horton House, Rugby. *Trans.* 1.

1836 †SHAW, ALEXANDER, Consulting Surgeon to the Middlesex Hospital; 136, Abbey road, Kilburn. C. 1842. S. 1843-4. V.P. 1851-2. T. 1858-60. *Referee*, 1842-3, 1846-50, 1855-7, 1865. *Lib. Com.* 1843. *Trans.* 4.

Elected

1859 SIBLEY, SEPTIMUS WILLIAM, 7, Harley street, Cavendish square. C. 1882. *Sci. Com.* 1863. *Trans.* 4.

1848 †SIEVEKING, EDWARD HENRY, M.D., *Librarian*, Physician-Extraordinary to H.M. the Queen; Physician-in-Ordinary to H.R.H. the Prince of Wales; Physician to St. Mary's Hospital; 17, Manchester square. C. 1859-60. S. 1861-3. V.P. 1873-4. L. 1881-2. *Referee*, 1855-8, 1864-72, 1875-80. *Sci. Com.* 1862. *Trans.* 2.

1842 †SIMON, JOHN, C.B., D.C.L., LL.D., F.R.S., Consulting Surgeon to St. Thomas's Hospital; 40, Kensington square. C. 1854-5. V.P. 1865. *Referee* 1851-3, 1866-81. *Trans.* 1.

1865 SIMS, J. MARION, M.D., Surgeon to the New York State Women's Hospital; 267, Madison Avenue, New York.

1857 SIORDET, JAMES LEWIS, M.B., Villa Preti, Mentone, Alpes Maritimes, France.

1882 SMITH, CHARLES JOHN, 54, Old Steyne, Brighton.

1879 SMITH, E. NOBLE, 24, Queen Anne street, Cavendish square.

1881 SMITH, EUSTACE, M.D., Physician to H.M. the King of the Belgians; Physician to the East London Hospital for Children, and to the City of London Hospital for Diseases of the Chest; 5, George street, Hanover square.

1872 SMITH, T. GILBART, M.A., M.D., Assistant-Physician to the London Hospital; Physician to the Royal Hospital for Diseases of the Chest, City road; 68, Harley street, Cavendish square. *Trans.* 1.

1866 SMITH, HEYWOOD, M.A. M.D. Oxon., Physician to the Hospital for Women; Physician to the British Lying-in Hospital; 18, Harley street, Cavendish square.

1838 †SMITH, SPENCER, Consulting Surgeon to St. Mary's Hospital; 92, Oxford terrace, Hyde Park. C. 1854. S. 1855-8. V.P. 1859-60. T. 1865. *Referee*, 1851-3, 1862-4, 1866-78. *Lib. Com.* 1847.

1863 SMITH, THOMAS, Surgeon to, and Lecturer on Clinical Surgery at, St. Bartholomew's Hospital; Surgeon to the Hospital for Sick Children; 5, Stratford place, Oxford street. S. 1870-2. C. 1875-76. *Referee*, 1873-4, 1880-82. *Sci. Com.* 1867. *Trans.* 3.

Elected

1847 SMITH, WILLIAM J., M.D., Consulting Physician to the Weymouth Infirmary; Greenhill, Weymouth, Dorsetshire.

1873 SMITH, W. JOHNSON, Surgeon to the Seamen's Hospital, Greenwich.

1874 *SMITH, WILLIAM ROBERT, M.D., F.R.S. Ed., Physician to the Dispensary, Cheltenham; 15, Imperial square, Cheltenham.

1868 SOLLY, SAMUEL EDWIN, Colorado Springs, Colorado, U.S.

1865 SOUTHEY, REGINALD, M.D., Physician to, and Lecturer on Forensic Medicine at, St. Bartholomew's Hospital; 6, Harley street, Cavendish square. C. 1881-2. *Referee*, 1873-80. *Trans* 1.

1844 SPACKMAN, FREDERICK R., M.D., Harpenden, St. Alban's.

1875 SPITTA, EDMUND J., Ivy House, Clapham Common, Surrey.

1851 †SPITTA, ROBERT JOHN, M.D. Lond., East Side, Clapham Common, Surrey. C. 1878-9. *Trans.* 1.

1854 STEVENS, HENRY, M.D., Inspector, Medical Department, Local Government Board; Greenford House, Sutton, Surrey.

1842 †STEWART, ALEXANDER PATRICK, M.D., Consulting Physician to the Middlesex Hospital; 75, Grosvenor street. C. 1856-7. L. 1863-8. V.P. 1871-2.

1859 STEWART, WILLIAM EDWARD, 16, Harley street, Cavendish square.

1879 *STIRLING, EDWARD CHARLES, late Assistant Surgeon and Lecturer on Physiology at St. George's Hospital; Adelaide, South Australia [care of T. Gemmell, Esq., 32, The Grove, Boltons, S.W.].

1856 STOCKER, ALONZO HENRY, M.D., Peckham House, Peckham,

1865 STOKES, WILLIAM, M.D., Examiner in Surgery, Queen's University, Ireland, and Surgeon to the Richmond Surgical Hospital; 5, Merrion square north, Dublin. *Trans.* 1.

1843 STORKS, ROBERT REEVE, Paris.

Elected

1858 †STREATFEILD, JOHN FREMLYN, Surgeon to the Royal London Ophthalmic Hospital, Moorfields; Professor of Clinical Ophthalmic Surgery in University College, and Senior Ophthalmic Surgeon to University College Hospital; 15, Upper Brook street, Grosvenor square. C. 1874-5. *Lib. Com.* 1867-8.

1871 STRONG, HENRY JOHN, M.D., 64, North End, Croydon.

1863 STURGES, OCTAVIUS, M.D., Physician to, and Lecturer on Medicine at, the Westminster Hospital; Assistant-Physician to the Hospital for Sick Children; 85, Wimpole street, Cavendish square. C. 1878-9. *Referee,* 1882.

1871 †SUTHERLAND, HENRY, M.D., Lecturer on Insanity at the Westminster Hospital; 6, Richmond terrace, Whitehall.

1860 SUTRO, SIGISMUND, M.D., Senior Physician to the German Hospital; 37A, Finsbury square.

1871 SUTTON, HENRY GAWEN, M.B., Physician to, and Lecturer on Pathology at, the London Hospital, and Physician to the City of London Hospital for Diseases of the Chest; 9, Finsbury square. *Trans.* 1.

1855 SUTTON, JOHN MAULE, M.D., Medical Officer of Health, Oldham; 244, Great Clowes street, Broughton, Manchester.

1861 *SWEETING, GEORGE BACON, King's Lynn, Norfolk.

1878 *SYMPSON, THOMAS, Surgeon to the Lincoln County Hospital; 3, James street, Lincoln.

1870 TAIT, LAWSON, Surgeon to the Birmingham and Midland Hospital for Women; 7, Great Charles street, Birmingham. *Trans.* 4.

1864 TAUSSIG, GABRIEL, M.D., 70, Piazza Barberini, Rome.

1875 TAY, WAREN, Surgeon to the London Hospital and Surgeon to the North Eastern Hospital for Children and the Hospital for Skin Diseases, Blackfriars; 4, Finsbury square.

1873 TAYLOR, FREDERICK, M.D., Assistant-Physician to, and Lecturer on Materia Medica at, Guy's Hospital; 11, St. Thomas's street, Southwark. *Trans.* 1.

Elected

1852 TAYLOR, ROBERT, 10, Portman square.

1845 †TAYLOR, THOMAS, Warwick House, 1, Warwick place, Grove End road, St. John's wood.

1859 TEGART, EDWARD, 49, Jermyn street, St. James's.

1874 THIN, GEORGE, M.D., 22, Queen Anne street, Cavendish square. *Trans.* 8.

1862 THOMPSON, EDMUND SYMES, M.D., Physician to the Hospital for Consumption, Brompton; Gresham Professor of Medicine; 33, Cavendish square. S. 1871-4. C. 1878-9. *Referee*, 1876-7. *Trans.* 1.

1857 THOMPSON, HENRY, M.D., Consulting Physician to the Middlesex Hospital; 53, Queen Anne street, Cavendish square.

1852 THOMPSON, SIR HENRY, Surgeon-Extraordinary to H.M. the King of the Belgians; Emeritus Professor of Clinical Surgery in University College, London; and Consulting Surgeon to University College Hospital; 35, Wimpole street, Cavendish square. C. 1869. *Trans.* 6.

1862 THOMPSON, REGINALD EDWARD, M.D., *Secretary*, Physician to the Hospital for Consumption, Brompton; 47, Park street, Grosvenor square. C. 1879. S. 1880-82. *Referee*, 1873-8. *Sci. Com.* 1867. *Trans.* 2.

1881 THOMSON, WILLIAM SINCLAIR, M.D., 40, Ladbroke grove, Kensington park gardens.

1876 THORNTON, JOHN KNOWSLEY, M.B., C.M., Surgeon to the Samaritan Free Hospital for Women and Children; 22, Portman street, Portman square. *Trans.* 2.

1875 TIBBITS, HERBERT, F.R.C.P. Ed., 68, Wimpole street.

1848 †TILT, EDWARD JOHN, M.D., Consulting Physician to the Farringdon General Dispensary and Lying-in Charity; 27, Seymour street, Portman square. *Referee*, 1874-81.

1880 TIVY, WILLIAM JAMES, 1, Tottenham place, Clifton, Bristol.

1872 TOMES, CHARLES S., B.A., F.R.S., Lecturer on Anatomy and Physiology at the Dental Hospital; 37, Cavendish square. *Lib. Com.* 1879.

1867 TONGE, MORRIS, M.D., Harrow-on-the-Hill, Middlesex.

Elected

1882 TOOTH, HOWARD HENRY, M.B., Casualty Physician to St. Bartholomew's Hospital; 25, Bernard street, Russell square.

1871 *TREND, THEOPHILUS W., M.D., Raeberry Lodge, Southampton.

1879 TREVES, FREDERICK, Assistant Surgeon to the London Hospital; 18, Gordon square.

1881 *TREVES, WILLIAM KNIGHT, Surgeon to the Royal Sea Bathing Infirmary for Scrofula; 31, Dalby square, Cliftonville, Margate.

1867 TROTTER, JOHN WILLIAM, Surgeon-Major, Coldstream Guards; Bossall Vicarage, York.

1859 TRUMAN, EDWIN THOMAS, Surgeon-Dentist in Ordinary to Her Majesty's Household; 23, Old Burlington street.

1864 TUFNELL, THOMAS JOLLIFFE, Consulting Surgeon to the City of Dublin Hospital; 58, Lower Mount street, Merrion square, Dublin. *Trans.* 1.

1862 TUKE, THOMAS HARRINGTON, M.D., Manor House, Chiswick, and 37, Albemarle street, Piccadilly.

1875 TURNER, FRANCIS CHARLEWOOD, M.A., M.D., Physician to the London Hospital; 15, Finsbury square.

1873 TURNER, GEORGE BROWN, M.D., San Remo, Italy.

1882 TURNER, GEORGE ROBERTSON, 9, Sussex gardens, Hyde park.

1881 TYSON, WILLIAM JOSEPH, M.D., Medical Officer of the Folkestone Infirmary; 10, Langhorne gardens, Folkestone.

1876 VENN, ALBERT JOHN, M.D., Obstetric Physician to the Metropolitan Free Hospital; Assistant Physician to the Victoria Hospital for Children; 8, Upper Brook street, Grosvenor square.

1870 VENNING, EDGCOMBE, 87, Sloane street.

1865 VERNON, BOWATER JOHN, Ophthalmic Surgeon to St. Bartholomew's Hospital and to the West London Hospital; 33, Curzon street, Mayfair.

Elected

1867 VINTRAS, ACHILLE, M.D., Physician to the French Embassy and to the French Hospital, Lisle street, Leicester square; 141, Regent street.

1828 VULPES, BENEDETTO, M.D., Physician to the Hospital of Aversa, and the Hospital of Incurables, Naples.

1854 WADDINGTON, EDWARD, Hamilton, Auckland, New Zealand.

1870 WADHAM, WILLIAM, M.D., Physician to St. George's Hospital; 14, Park lane.

1864 WAITE, CHARLES DERBY, M.B., Senior Physician to the Westminster General Dispensary; 3, Old Burlington street.

1868 *WALKER, ROBERT, L.R.C.P. Edinb., Surgeon to the Carlisle Dispensary; 2, Portland square, Carlisle.

1867 *WALLIS, GEORGE, Surgeon to Addenbrooke's Hospital, Corpus Buildings, Cambridge.

1873 WALSHAM, WILLIAM JOHNSON, C.M., Assistant Surgeon to, and Demonstrator of Anatomy and Operative Surgery at, St. Bartholomew's Hospital; Surgeon to the Metropolitan Free Hospital and to the Royal Hospital for Diseases of the Chest, City Road; 27, Weymouth street, Portland place. *Lib. Com.* 1882. *Trans.* 2.

1852 WALSHE, WALTER HAYLE, M.D., Emeritus Professor of the Principles and Practice of Medicine, University College, London; Consulting Physician to the Hospital for Consumption; 41, Hyde park square. C. 1872. *Trans.* 1.

1851 †WALTON, HAYNES, Senior Surgeon to St. Mary's Hospital, 1, Brook street, Grosvenor square. *Trans.* 1. *Pro.* 1.

1852 WANE, DANIEL, M.D., 20, Grafton street, Berkeley square.

1821 WARD, WILLIAM TILLEARD, Tilleards, Stanhope, Canada.

1858 WARDELL, JOHN RICHARD, M.D., Calverley park, Tunbridge Wells.

1846 WARE, JAMES THOMAS, Tilford House, near Farnham, Surrey.

1818 WARE, JOHN, Clifton Down, near Bristol.

1866 WARING, EDWARD JOHN, C.I.E., M.D., 49, Clifton gardens, Maida vale. *Referee*, 1881-2.

Elected

1877 WARNER, FRANCIS, M.D., Assistant Physician to the London Hospital and to the East London Hospital for Children; 24, Harley street, Cavendish square.

1861 WATERS, A. T. HOUGHTON, M.D., Physician to the Royal Infirmary, and Lecturer on the Principles and Practice of Medicine, in the Liverpool Royal Infirmary School of Medicine; 69, Bedford street, Liverpool. *Trans.* 3.

1879 WATERS, JOHN HENRY, M.D., C.M., 101, Jermyn street.

1878 WATNEY, HERBERT, M.D., Assistant Physician to St. George's Hospital; 1, Wilton crescent, Belgrave square.

1837 †WATSON, SIR THOMAS, Bart., M.D., D.C.L., F.R.S., Physician-in-Ordinary to H.M. the Queen; Consulting Physician to King's College Hospital; 16, Henrietta street, Cavendish square. C. 1840-1, 1852. V.P. 1845-6. *Referee*, 1842-5, 1847-9.

1861 †WATSON, WILLIAM SPENCER, M.B., Surgeon to the Great Northern Hospital; Surgeon to the Royal South London Ophthalmic Hospital; 7, Henrietta street, Cavendish square. *Trans.* 1.

1879 WATTEVILLE, ARMAND DE, M.B., B.S., Medical Electrician to St. Mary's Hospital; 9, Wimpole street, Cavendish square.

1854 WEBB, WILLIAM, M.D., Gilkin View House, Wirksworth, Derbyshire.

1840 WEBB, WILLIAM WOODHAM, M.D., 82, Avenue des Termes, Paris.

1842 †WEBER, FREDERIC, M.D., 44, Green street, Park lane. C. 1857. V.P. 1865.

1857 WEBER, HERMANN, M.D., Physician to the German Hospital; 10, Grosvenor street, Grosvenor square. C. 1874-5. *Referee*, 1869-73, 1878-82. *Lib. Com.* 1864-73. *Trans.* 6.

1844 †WEGG, WILLIAM, M.D., 15, Hertford street, Mayfair. L. 1854-8. C. 1861-2. T. 1873-80. *Lib. Com.* 1851-3.

1878 WEISS, HUBERT FOVEAUX, 30A, George street, Hanover square.

Elected

1874 WELLS, HARRY, M.D., British Vice-Consulate, Gualeguay-chu, Entre Rios, Argentine Confederation.

1854 WELLS, THOMAS SPENCER, Surgeon-in-Ordinary to H.M.'s Household; Consulting Surgeon to the Samaritan Free Hospital for Women and Children; 3, Upper Grosvenor street. C. 1870. V.P. 1881. *Trans.* 12. *Pro.* 1.

1842 †WEST, CHARLES, M.D., Corresponding Member of the Academy of Medicine of Paris; 29, Promenade des Anglais, Nice, Alpes Maritimes, France, and 2, Bolton row, Mayfair, W. C. 1855-6. V.P. 1863. P. 1877-8. *Referee*, 1848-54, 1857-62, 1864-76, 1880. *Sci. Com.* 1863. *Lib. Com.* 1844-7, 1851. *Trans.* 2.

1877 WEST, SAMUEL, M.B., Casualty Physician and Medical Tutor at St. Bartholomew's Hospital; Assistant Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; 15, Wimpole street, Cavendish square.

1882 WHARRY, CHARLES JOHN, M.D., Resident Superintendent, Government Civil Hospital, Hong Kong.

1881 WHARRY, ROBERT, M.D., 6, Gordon square.

1878 WHARTON, HENRY THORNTON, M.A., Surgeon to the Kilburn Dispensary; 39, St. George's road, Kilburn.

1828 WHATLEY, JOHN, M.D.

1875 WHIPHAM, THOMAS TILLYER, M.B., Physician to, and Lecturer on Clinical Medicine at, St. George's Hospital; 11, Grosvenor street, Grosvenor square.

1849 WHITE, JOHN.

1881 WHITE, WILLIAM HALE, M.D., Demonstrator of Anatomy at Guy's Hospital; 4, St. Thomas's street, Southwark.

1881 *WHITEHEAD, WALTER, F.R.S. Ed., Surgeon to the Manchester Royal Infirmary; 24, St. Ann's square, Manchester.

1877 WHITMORE, WILLIAM TICKLE, 7, Arlington street, Piccadilly.

1852 WIBLIN, JOHN, M.D., Medical Inspector of Emigrants and Recruits; Southampton. *Trans.* 1.

1870 *WILKIN, JOHN F., M.D. and M.C., New Beckenham, Kent.

Elected

1837 WILKS, GEORGE AUGUSTUS FREDERICK, M.D., Stanbury, Torquay.

1863 WILKS, SAMUEL, M.D., F.R.S., Physician to, and Lecturer on Medicine at, Guy's Hospital; Physician in Ordinary to their Royal Highnesses the Duke and Duchess of Connaught; 72, Grosvenor street, Grosvenor square. *Referee*, 1872-81. *Sci. Com.* 1.

1865 †WILLETT, ALFRED, Surgeon to St. Bartholomew's Hospital; Surgeon to St. Luke's Hospital; 36, Wimpole street, Cavendish square. C. 1880-81. *Referee*, 1882. *Trans.* 1.

1864 WILLETT, EDMUND SPARSHALL, M.D., Resident Physician, Wyke House, Isleworth, Middlesex.

1840 †WILLIAMS, CHARLES JAMES BLASIUS, M.D., F.R.S., Physician-Extraordinary to H.M. the Queen; Consulting Physician to the Hospital for Consumption, Brompton [47, Upper Brook street, Grosvenor square]. C. 1849-50. V.P. 1860-1. P. 1873-4. *Referee*, 1843-4. *Sci. Com.* 1862. *Trans.* 1.

1859 *WILLIAMS, CHARLES, Surgeon to the Norfolk and Norwich Hospital; 9, Prince of Wales road, Norwich.

1866 WILLIAMS, CHARLES THEODORE, M.A., M.D., Physician to the Hospital for Consumption and Diseases of the Chest, Brompton; 47, Upper Brook street, Grosvenor square. *Lib. Com.* 1880-2. *Trans.* 3.

1881 WILLIAMS, DAWSON, M.B., 4, Oxford and Cambridge Mansions, Marylebone road.

1872 WILLIAMS, JOHN, M.D., Assistant Obstetric Physician to University College Hospital; 28, Harley street, Cavendish square. *Referee*, 1878-82. *Lib. Com.* 1876-82.

1868 WILLIAMS, WILLIAM RHYS, M.D., Commissioner in Lunacy; 19, Whitehall place.

1839 †WILSON, SIR ERASMUS, F.R.S., late Professor of Dermatology, Royal College of Surgeons of England; 17, Henrietta street, Cavendish square. C. 1877. *Lib. Com.* 1845. *Trans.* 2.

1863 WILSON, ROBERT JAMES, F.R.C.P. Edin., 7, Warrior square, St. Leonard's-on-Sea, Sussex.

Elected

1850 *WISE, ROBERT STANTON, M.D., Consulting Physician to the Southam Eye and Ear Infirmary; Banbury, Oxfordshire.

1825 WISE, THOMAS ALEXANDER, M.D., Inchrye Abbey, Newborough, Fife, N.B.

1879 WOAKES, EDWARD, M.D., Senior Aural Surgeon to the London Hospital; 57, Harley street, Cavendish square.

1851 †WOOD, JOHN, F.R.S., Professor of Clinical Surgery in King's College, London, and Senior Surgeon to King's College Hospital; Examiner in Surgery in the University of London; 61, Wimpole street, Cavendish square. C. 1867-8. V.P. 1877-8. *Referee*, 1871-6, 1880-82. *Lib. Com.* 1866. *Trans.* 3.

1848 †WOOD, WILLIAM, M.D., Physician to St. Luke's Hospital for Lunatics; 99, Harley street, Cavendish square. C. 1867-8. V.P. 1877-8.

1881 WOODMAN, SAMUEL, Consulting Surgeon to the Ramsgate and St. Lawrence Royal Dispensary; 5, Prospect terrace, Ramsgate.

1879 WOODWARD, G. P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1842 WORTHINGTON, WILLIAM COLLINS, London road, Lowestoft, Suffolk. *Trans.* 3.

1865 WOTTON, HENRY, M.D., 62, Bedford gardens, Kensington.

1878 YEO, GERALD F., M.D., M.Ch., Professor of Physiology in King's College, London; King's College, Strand.

[It is particularly requested that any change of Title, Appointment, or Residence, may be communicated to the Secretaries before the 1st of October in each year, in order that the List may be made as correct as possible.]

HONORARY FELLOWS.

(Limited to Twelve.)

Elected

1847 CHADWICK, EDWIN, C.B., Corresponding Member of the Academy of Moral and Political Sciences of the Institute of France; Park Cottage, East Sheen.

1857 FARR, WILLIAM, M.D., C.B., D.C.L., F.R.S., 78, Portsdown road, Maida hill.

1868 HOOKER, SIR JOSEPH DALTON, M.D., C.B., K.C.S.I., D.C.L., LL.D., F.R.S., Member of the Senate of the University of London, Director of the Royal Botanic Gardens, Kew; Corresponding Member of the Academy of Sciences of the Institute of France; Royal Gardens, Kew.

1868 HUXLEY, THOMAS HENRY, LL.D., F.R.S., Professor of Natural History in the Royal School of Mines; Secretary to the Royal Society; Corresponding Member of the Academies of Sciences of St. Petersburg, Berlin, Dresden, &c.; 4, Marlborough place, St. John's wood.

1878 LUBBOCK, SIR JOHN, Bart., M.P., D.C.L., LL.D., F.R.S., High Elms, Bromley, Kent.

1847 OWEN, RICHARD, C.B., D.C.L., LL.D., F.R.S., Superintendent of the Natural History Departments in the British Museum; Foreign Associate of the Academy of Sciences of the Institute of France; Sheen Lodge, Mortlake.

Elected

1873 STOKES, GEORGE GABRIEL, M.A., D.C.L., LL.D., Lucasian Professor of Mathematics in the University of Cambridge; Secretary to the Royal Society, &c.; Lensfield Cottage, Cambridge.

1868 TYNDALL, JOHN, D.C.L., LL.D., F.R.S., Professor of Natural Philosophy in the Royal Institution; Corresponding Member of the Academies and Societies of Sciences of Göttingen, Haarlem, Geneva, &c.; Royal Institution, Albemarle street, Piccadilly.

FOREIGN HONORARY FELLOWS.

(Limited to Twenty.)

Elected

1878 Baccelli, Guido, M.D., Professor of Medicine at Rome.

1876 Billroth, Theodor, M.D., Professor of Surgery in the University of Vienna; Vienna.

1864 Donders, Franz Cornelius, M.D., LL.D., Professor of Physiology and Ophthalmology at the University of Utrecht.

1876 Edwards, H. Milne, M.D., Member of the Institute of France, and of the Academy of Medicine; Dean of the Faculty of Sciences and Professor at the Museum of Natural History of Paris; 57, Rue Cuvier, Paris.

1835 Ekströmer, Carl Johan, M.D., C.M., K.P.S., and W., Physician to the King of Sweden; President of the College of Health, and Director-General of Hospitals; Stockholm.

1868 Gross, Samuel D., M.D., F.C.P. Philad., D.C.L. Oxon., LL.D. Camb., Emeritus Professor of Surgery in the Jefferson Medical College of Philadelphia.

1878 Gueneau de Mussy, Noel, M.D., Member of the Academy of Medicine; Physician to the Hôtel Dieu; 4, Rue St. Arnaud, Paris.

1866 Hannover, Adolph, M.D., Professor at Copenhagen.

1873 Helmholtz, Hermann Ludwig Ferdinand, Professor of Physics and Physiological Optics; Berlin.

1859 Henle, J., M.D., Professor of Anatomy at Göttingen.

1873 Hofmann, A. W., LL.D., Ph.D., Professor of Chemistry, Berlin.

Elected

1868 KÖLLIKER, ALBERT, Professor of Anatomy in the University of Würzburg.

1856 LANGENBECK, BERNHARD, M.D., late Professor of Surgery in the University of Berlin.

1868 LARREY, HIPPOLYTE BARON, Member of the Institute of France; Inspector of the "Service de Santé Militaire," and Member of the "Conseil de Santé des Armées;" Commander of the Legion of Honour, &c.; Rue de Lille, 91, Paris.

1878 SCANZONI, FRIEDREICH WILHELM VON, Royal Bavarian Privy Councillor, and Professor of Medicine in the University of Würzburg.

1856 VIRCHOW, RUDOLPH, M.D., Professor of Pathological Anatomy in the University of Berlin; Corresponding Member of the Academy of Sciences of the Institute of France; Berlin.

LIST OF RESIDENT FELLOWS

ARRANGED ACCORDING TO

DATE OF ELECTION.

1819 Jas. M. Arnott, F.R.S.
1828 Cæsar H. Hawkins, F.R.S.
1833 Sir George Burrows, Bt., M.D., F.R.S.
Thomas A. Barker, M.D.
1835 Richard Quain, F.R.S.
Thomas A. Nelson, M.D.
1836 Alexander Shaw.
J. George French.
1837 Sir Thomas Watson, Bt., M.D., F.R.S.
Thomas Blizard Curling, F.R.S.
George Busk, F.R.S.
Thomas William Jones, M.D.
1838 Charles Hawkins.
Henry Spencer Smith.
1839 T. Graham Balfour, M.D., F.R.S.
Sir W. J. Erasmus Wilson, F.R.S.
Fred. Le Gros Clark, F.R.S.
James Dixon.
1840 Chas. J. B. Williams, M.D., F.R.S.
Charles Hutton, M.D.
Samuel A. Lane.
Sir James Paget, Bt., F.R.S.
1841 Henry A. Pitman, M.D.
William Bowman, F.R.S.
John Parkin, M.D.
Paul Jackson.
1842 Charles West, M.D.
Frederic Weber, M.D.
Alex. P. Stewart, M.D.
John Simon, C.B., F.R.S.
John Erichsen, F.R.S.
Oscar M. P. Clayton.
1843 Robert Greenhalgh, M.D.
1843 Prescott G. Hewett, F.R.S.
Henry Lee.
Wm. White Cooper.
Luther Holden.
Edward Newton.
1844 Arthur Farre, M.D., F.R.S.
William Wegg, M.D.
Thomas King Chambers, M.D.
Nathaniel Montefiore.
Edwin Humby.
1845 Samuel Cartwright.
George D. Pollock.
Thomas Taylor.
Edwin Saunders.
William Oliver Chalk.
Edward U. Berry.
Benjamin Ridge, M.D.
1846 John A. Bostock.
Barnard Wight Holt.
Carsten Holthouse.
1847 Andrew Whyte Barclay, M.D.
W. H. O. Sankey, M.D.
George Johnson, M.D., F.R.S.
George Critchett.
1848 Edward H Sieveking, M.D.
Edward Ballard, M.D.
William Wood, M.D.
Thomas Hawksley, M.D.
Edward John Tilt, M.D.
John Clarke, M.D.
John Gay.
John Gregory Forbes.
1849 Hugh J. Sanderson, M.D.
C. H. F. Routh, M.D.
Edmund L. Birkett, M.D.

1849 George T. Fincham, M.D.
Sir William W. Gull, Bt., M.D., F.R.S.
1850 Richard Quain, M.D., F.R.S.
George Roper, M.D.
1851 Sir Wm. Jenner, Bt., M.D., F.R.S.
H. Haynes Walton.
John Birkett.
John A. Kingdon.
Peter Y. Gowlland.
Frederic John Mouat, M.D.
John Marshall, F.R.S.
John Wood, F.R.S.
Bernard E. Brodhurst.
Robert J. Spitta, M.D.
George Gaskoin.
1852 C. Bland Radcliffe, M.D.
Daniel Wane, M.D.
Walter H. Walshe, M.D.
William Adams.
John Cooper Forster.
Sir Henry Thompson.
Robert Taylor.
Richard Phillips.
1853 Robert Brudenell Carter.
1854 Alfred Baring Garrod, M.D., F.R.S.
Samuel O. Habershon, M.D.
Thomas Spencer Wells.
1855 W. M. Graily Hewitt, M.D.
J. Burdon Sanderson, M.D., F.R.S.
J. Russell Reynolds, M.D., F.R.S.
James Robert Lane.
Walter John Bryant, M.D.
1856 Charles J. Hare, M.D.
William Bird.
Jonathan Hutchinson, F.R.S.
Timothy Holmes.
Alonzo H. Stocker, M.D.
1857 William Overend Priestley, M.D.
George Harley, M.D., F.R.S.
Henry Thompson, M.D.
Hermann Weber, M.D.
George Owen Rees, M.D., F.R.S.
John Whitaker Hulke, F.R.S.
John Morgan.
Henry Cooper Rose, M.D.
Henry Walter Kiallmark.
1858 Fred. George Reed, M.D.
William Chapman Begley, M.D.
John William Ogle, M.D.
Wilson Fox, M.D., F.R.S.
John Fremlyn Streatfield.
Francis Harris, M.D.
1859 Wm. Howship Dickinson, M.D.
1859 William Scovell Savory, F.R.S.
Edwin Thomas Truman.
Francis Hird.
Richard Barwell.
Edward Tegart.
Septimus William Sibley.
William E. Stewart.
1860 Andrew Clark, M.D.
John Maclean, M.D.
Sigismund Sutro, M.D.
William Ogle, M.D.
Thomas Bryant.
John Couper.
Henry Howard Hayward.
1861 Robert Barnes, M.D.
William Spencer Watson.
William Henry Holman.
1862 James Andrew, M.D.
Lionel Smith Beale, M.B., F.R.S.
Thomas H. Tuke, M.D.
Samuel Crompton, M.D.
Edmund Symes Thompson, M.D.
Reginald Edward Thompson, M.D
William Henry Brace, M.D.
George Cowell.
M. Berkeley Hill.
1863 Octavius Sturges, M.D.
John Langdon H. Down, M.D.
Samuel Wilks, M.D., F.R.S.
Samuel Fenwick, M.D.
Julius Althaus, M.D.
Sydney Ringer, M.D.
Thomas Smith.
Arthur B. R. Myers.
Arthur E. Durham.
William Sedgwick.
1864 Charles Hilton Fagge, M.D.
George Buchanan, M.D., F.R.S.
Charles Derby Waite, M.B.
John Harley, M.D.
Walter John Coulson.
Thomas William Nunn.
Francis Mason.
Jos. Gillman Barratt, M.D.
1865 Charles Robert Drysdale, M.D.
James Edward Pollock, M.D.
William Cholmeley, M.D.
Reginald Southey, M.D.
George Fielding Blandford, M.D.
Dyce Duckworth, M.D.
Frederick W. Pavy, M.D., F.R.S.
William Morrant Baker.
John Langton.
Frederick James Gant.

1865 Alfred Willett.
Bowater John Vernon.
Alfred Cooper.
Christopher Heath.
Henry Wotton.
1866 Thomas Fitzpatrick, M.D.
Samuel Jones Gee, M.D.
Charles Theodore Williams, M.D.
Heywood Smith, M.D.
John Crockett Fish, M.D.
William Selby Church, M.D.
Edward John Waring, M.D.
William Fairlie Clarke, M.D.
Philip H. Harper.
1867 William Henry Day, M.D.
Achille Vintras, M.D.
Richard Douglas Powell, M.D.
F. Howard Marsh.
Henry Power.
Sir William MacCormac.
Thomas Pickering Pick.
John Astley Bloxam.
Charles Arthur Aikin.
Samuel Hill, M.D.
Colomiati Meredyth, M.D.
1868 H. Charlton Bastian, M.D., F.R.S.
William Henry Broadbent, M.D.
Thomas Buzzard, M.D.
John Cavafy, M.D.
Walter Butler Cheadle, M.D.
John Cockle, M.D.
William Johnston, M.D.
T. Henry Green, M.D.
William Rhys Williams, M.D.
Walter Moxon, M.D.
William Chapman Grigg, M.D.
John Croft.
George Eastes.
William Henry Freeman.
1869 Joseph Frank Payne, M.D.
Arthur E. Sansom, M.D.
John Wickham Legg, M.D.
Charles Elam, M.D.
Thomas Laurence Read.
1870 Alfred Meadows, M.D.
William Wadham, M.D.
J. Warrington Haward.
Edgcombe Venning.
Clement Godson, M.D.
1871 William Cayley, M.D.
Charles Henry Ralfe, M.D.
Arthur Julius Pollock, M.D.
Thomas L. Brunton, M.D., F.R.S.
Henry Gawen Sutton, M.D.
1871 J. Hughlings Jackson, M.D., F.R.S.
Henry Sutherland, M.D.
George Vivian Poore, M.D.
Walter Rivington.
Marcus Beck.
Edward Bellamy.
William F. Butt.
Benjamin Duke.
1872 Gilbart Smith, M.D.
Thomas B. Christie, M.D.
George B. Brodie, M.D.
John Williams, M.D.
Sir J. Fayrer, M.D., F.R.S.
Charles S. Tomes, B.A., F.R.S.
William Bartlett Dalby.
1873 William Miller Ord, M.D.
Frederick Taylor, M.D.
Norman Moore, M.D.
John Curnow, M.D.
William R. Gowers, M.D.
William Guyer Hunter, M.D.
Charles Creighton, M.D.
Jeremiah McCarthy.
Wm. Johnson Smith.
Robert William Parker.
Alex. O. McKellar.
Henry T. Butlin.
Charles Higgens.
William J. Walsham.
Edward Milner.
1874 Alfred Lewis Galabin, M.D.
George Thin, M.D.
Alfred B. Duffin, M.D.
James H. Aveling, M.D.
Evan B. Baxter, M.D.
John M. Bruce, M.D.
Henry Morris.
William Laidlaw Purves.
William Harrison Cripps.
Henry G. Howse.
Herbert William Page.
Frederic Durham.
John J. Merriman.
1875 Thomas T. Whipham, M.B.
Francis Charlewood Turner, M.D.
William K. Murphy, M.D.
Herbert Tibbits, F.R.C.P. Ed.
Robert Hunter Semple, M.D.
Thomas Crawford Hayes, M.D.
Charles Henry Carter, M.D
Fletcher Beach, M.B.
Samuel Osborn.
Waren Tay.
Edmund J. Spitta.

1876 Thomas Barlow, M.D.
John C. Bucknill, M.D., F.R.S.
Wm. Lewis Dudley, M.D.
Albert J. Venn, M.D.
John Knowsley Thornton.
Charles Macnamara.
John N. C. Davies-Colley.
1877 Felix Semon, M.D.
Sidney Coupland, M.D.
Francis Warner, M.D.
T. Cranstoun Charles, M.D.
William Ewart, M.B.
Alfred Pearce Gould.
J. Rickman Godlee.
Alban H. G. Doran.
George Ernest Herman, M.B.
Samuel West, M.B.
John Abercrombie, M.B.
J. Matthews Duncan, M.D., F.R.S. Ed.
Henry de Fonmartin, M.D.
George Allan Heron, M.D.
Joseph A. Ormerod, M.B.
P. Henry Pye-Smith, M.D.
Edward Nettleship.
William Henry Bennett.
Benj. T. Lowne.
William T. Whitmore.
1878 Jas. Crichton Browne, M.D.
Fred. T. Roberts, M.D.
Joseph Lister, F.R.S.
Clinton T. Dent.
John H. Morgan.
Walter Pye.
Gerald F. Yeo, M.D.
Wm. J. Vereker Bindon, M.D.
Donald W. Charles Hood, M.B.
Henry Gervis, M.D.
Herbert Watney, M.D.
Richard Davy.
Hubert Foveaux Weiss.
Henry Thornton Wharton.
1879 Alfred Sangster, M.B.
Edward Woakes, M.D.
Armand de Watteville.
Malcolm A. Morris.
A. E. Cumberbatch.
Edmund Owen.
Arthur E. J. Barker.
Frederick Treves.
Horatio Donkin, M.B.
Thomas John Maclagan, M.D.
David White Finlay, M.D.
Andrew Clark.

1879 S. Hamilton Cartwright.
John H. Waters, M.D.
Francis Henry Champneys, M.D.
William Watson Cheyne.
William Munk, M.D.
George Henry Savage, M.D.
H. H. Clutton, M.A.
Frederic S. Eve.
E. Noble Smith.
William Henry Allchin, M.B
F. G. Dawtrey Drewitt, M.B.
1880 Robert Alex. Gibbons, M.D.
David Ferrier, M.D., F.R.S.
Vincent Dormer Harris, M.D.
Edmund D. Maddick.
Jas. John MacWhirter Dunbar, M.B.
James William Browne, M.B.
William Appleton Meredith, M.B.
Alexander Hughes Bennett, M.D.
Malcolm Macdonald McHardy.
A. Boyce Barrow.
William Murrell, M.D.
Charles Cyril Hicks, M.D.
Bernard O'Connor, A.B., M.D.
Leslie Ogilvie, M.B.
George Lockwood Laycock, M.B.
George Ogilvie, M.B.
Charles Edward Beevor, M.B.
Thomas Colcott Fox, M.B.
George Henry Makins.
1881 Francis de Havilland Hall, M.D.
Robert Wharry, M.D.
Cecil Yates Biss, M.B.
Richard Clement Lucas.
Stephen Mackenzie, M.D.
James Anderson, M.D.
William Hale White, M.D.
Eustace Smith, M.D.
George Rice, M.B.
William Sinclair Thomson, M.D.
Percy Kidd, M.D.
Oswald A. Browne, M.A.
Audley Cecil Buller.
W. Bruce Clarke, M.B.
Dawson Williams, M.B.
George Lindsay Johnson, M.A.
Henry Edward Juler.
Jonathan F. C. H. Macready.
Henry Howard, M.B.
Samuel Nall, M.B.
C. B. Lockwood.
1882 Philip J. Hensley, M.D.
Ernest Clarke.
Isidore I. Lyons.

1882 John Barclay Scriven.
Yelverton Dawson, M.D.
Charles L. Roy, M.D.
George Robertson Turner.
David Alexander King.
Laurence Humphry, M.B.
Howard Henry Tooth, M.B.
Herbert Isambard Owen.

1882 Charles R. B. Keetley.
Joseph Mills.
A. T. Myers, M.D.
Anthony A. Bowlby.
Amand J. McC. Routh, M.B.
Seymour J. Sharkey, M.B.
William Lang.

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The Abstracts of the papers read will be furnished to the Journals as heretofore.

ON GANGRENOUS ERUPTIONS

IN

CONNECTION WITH VACCINATION AND CHICKEN-POX.

BY

JONATHAN HUTCHINSON, F.R.S.,

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Received October 7th—Read October 25th, 1881.

I HAD the honour to exhibit before the Society about two years ago, the body of an infant who had died of a severe gangrenous eruption, which had followed vaccination.

I believed that it was an example of "Vaccinia gangrenosa," *i.e.* a vaccinia eruption which had taken on a gangrenous form. I now purpose to bring before the Society the details of the case, and also to give some account of an allied and much more common malady to which for many years past I have applied the name "Varicella gangrenosa."

Details of Case in which Death followed from a Gangrenous Eruption after Vaccination.

A male infant, aged about three months, and in excellent health, was vaccinated on November 11th, 1879, at a district

office. The vaccination was from arm to arm, and the vaccinifer appeared to be in perfect health. Four others were vaccinated from the same source at the same time, and nothing unusual happened. On the eighth day after the vaccination the child was brought again to the station for examination. It had four pearly vesicles on the arm, which showed nothing unusual, but its body and limbs were covered by an eruption of a peculiar character. This eruption, which had been coming out for a day or two, was described by my informant, the vaccinator, as looking like smallpox. He said that the papules were distinctly shotty, and, believing it to be a case in which variola had been contracted prior to vaccination, he instructed the mother to take the child home and on no account to bring it to the station again. Four days later he visited the child at its home and found the pustules much developed, whilst in many of them gangrene was commencing. He now advised that the attendance of the parish medical officer should be obtained. This was not done, and between this date and that of the child's death no medical man saw it. The death occurred on November 30th, twenty days after vaccination. Under the peculiar circumstances an inquest became necessary, and an opinion having been expressed that the case was one of vaccinal syphilis, I was requested by the coroner to examine the body and make a report.

The state of the child's skin after death may be realised with tolerable accuracy by the portrait (see Plate I). The vaccination spots were covered with scabs, and there was a certain amount of congestion around them, but none of them were in the least indurated.

On the scalp and face there were spots and patches ranging in size from a shot to a shilling, some of them simply congested or scabbed, but others showing a central area of gangrenous skin. On the trunk, both back and front, there were similar spots with some also much larger. All the larger ones showed a rim of deep ulceration which surrounded a central slough of black skin. From a few

the slough had separated, a deep ulcer remaining. But few of the ulcers were quite round, and in many instances the shape was irregular, as if several spots had coalesced. The eruption occurred on both sides of the body alike, but as regards the larger eschars, it was by no means arranged in exact symmetry. The back was very severely affected, and the lower part of the abdomen and upper parts of the thighs. There were some large eschars near the knees, but the lower halves of the legs and the feet, and the whole of both upper extremities below the elbows, were almost wholly free.

A post-mortem examination of the body was made for me by Dr. Barlow, who reported that the viscera were free from disease and that the child appeared to have been in a state of good nutrition.

So far as I am aware this is the first case of gangrenous eruption after vaccination which has been recorded, but that a general exanthematous rash does sometimes follow at the end of the first week after vaccination has long been well known. To this eruption the term vaccinial exanthem has been given. Several somewhat different forms of eruption have been recognised in this connection. Sometimes the rash is simply erythematous, at others papular, and in a few it closely resembles varicella. It has been described in detail by Mr. Ceely and others. I believe that the case just narrated was an example of vaccinia which became gangrenous, and it is of interest to note that the primary lesion of the skin was a hard papule with an umbilicus, and so closely resembling variola that it was taken for that disease by the surgeon who saw the case.

In a paper written some years ago, I attempted to draw attention to the fact that a peculiar form of prurigo, which is a common consequence of varicella, occurs also sometimes after vaccination, and probably in connection with the vaccinia rash. Thus it would appear that there are certain features of similarity as regards the pathological possibilities of vaccinia and varicella, and to these

I wish now to add the fact that they are both liable to become gangrenous and to end fatally.

Before I pass to the subject of varicella gangrenosa, I wish to mention another case of gangrenous vaccinia which has occurred in Dublin since my communication to this Society in November, 1879. The case occurred under the care of Mr. William Stokes of the Richmond Hospital, and I am indebted to him not only for particulars of the case, and permission to make use of them, but also for being able to bring before the Society an excellent portrait of the patient. In Mr. Stokes' case the gangrenous patches were much larger than any which occurred in mine. Those on the buttocks were several inches in length. As in my case they are irregular in shape, and evidently produced by the confluence of groups of papules. It is unfortunate that in this case there is uncertainty as to the date of vaccination and consequently as to the length of the interval before the appearance of the eruption.

Neither in this case nor in my own were the vaccination sores themselves attacked by gangrene; it was the general eruption only which was so affected. The full details of Mr. Stokes' case have been published by him in the 'Dublin Journal of Medical Science' for June, 1880.

Varicella Gangrenosa.

The earlier cases which came under my notice were mostly in the late stages of the disease, and none were so characteristic as in a later one of which the portrait is produced. In this case the patient was under the care of Dr. David Lees at the Children's Hospital, Great Ormond Street, who kindly procured me an opportunity for seeing it.

In my own cases the patients usually came under observation in the surgical practice at the London Hospital, on account of deep gangrenous ulcers in the flexures, and with a few scattered spots on the skin. It was by the history only that I was enabled to connect such cases with chicken-pox.

I am sure that I have seen five or six of this kind, but

I regret that I have not preserved notes of any in a form which would justify my producing them to this Society. I recorded in the 'Ophthalmic Hospital Reports,' vol. vi, 146, a case in which a young child after an eruption much like chicken-pox had a double irido-choroiditis, and lost both eyes. A few years later a case similar to this came under my care at Moorfields, the child being brought there on account of double iritis. The child had several scattered spots of gangrene where the vesicles had been. I suggested gangrenous varicella, but was unable to carry the proof further as there was no evidence to show that the child had been exposed to contagion.

In a lecture on some of the peculiarities of varicella, delivered many years ago, I mentioned "a form of ulcerating chicken-pox which in syphilis finds its homologue in rupia." I then quoted a passage from Trousseau, who long before had described an epidemic of chicken-pox, in which the eruption had been protracted for six weeks or two months, and had produced ulcerations like those of pemphigus.

There are in Guy's Hospital Museum excellent wax casts from two cases which I should diagnose as gangrenous varicella. They are classed under the name of rupia escharotica.[1] In the description of one of the models it is said "the earliest stage of the affection is seen in certain vesicles ranging in size from a pin's head to a split pea. Some of them are flattened and have red areola round them. Subsequently the vesicles became larger and ulcerated." It is stated in this case that the child recovered in a fortnight without having taken any medicine, a fact strongly at variance with the usual history of pemphigus. No one who has examined these models and read Dr. Fagge's descriptions, and who is acquainted with Dr. Whitley Stokes' description seventy years ago of what he called "Pemphigus gangrenosus," can doubt for a moment that the "Rupia escharotica" of Guy's and the pemphigus gangrenosus of Irish observers are one and the same malady. I hope to make it scarcely less clear that they are both of them really forms of varicella.

[1] Models 206 to 209.

Dr. Whitley Stokes, in 1807, wrote in the 'Dublin Medical and Physical Essays' a paper on "An Eruptive Disease of Children." He proposed for it the name of "Pemphigus gangrenosus" or "White blisters." He described it as very severe and frequently fatal, and said that it was well known in many parts of Ireland. It is not improbable that several different maladies contributed to his description, but that his attention had been attracted to cases of gangrenous chicken-pox is, I think, almost certain. He said that the eruption usually developed itself during perfect health, and that it occurred by preference to strong children. "One or more vesicles appear, mostly larger than the most distinct smallpox; these increase for two or three days, burst, and discharge a thin fluid and a disagreeable smell." The febrile disturbance which accompanies it he believed to be induced by the irritation of the eruption. Death usually occurred about the tenth or twelfth day, and in those who recovered relapses were common. Most of the patients were between the ages of three months and four years, but it had been observed as late as nine years. The flexures of the joints and behind the ears were frequently affected by gangrenous ulcers, and gangrenous spots were the characteristic of the disease.

Dr. Whitley Stokes distinguished the disease from chicken-pox, but he evidently came very near to the recognition of what I believe to have been its true character. In speaking of diagnosis, he wrote: "On the other hand, the swine-pox (varicella) resembles this disease in its first stage; but the fever rarely precedes the eruption in 'white blisters,' and the pustules of varicella dry quickly."

If we put aside the assumption that in all cases the vesicles of varicella dry quickly, we have no point left for differential diagnosis except the occurrence in varicella of fever before the eruption, and it is now well known that this is often, if not usually, so slight as to be easily overlooked.

Dr. Stokes mentioned that in some cases the eye

is inflamed, a fact which, as already mentioned, has fallen under my own notice. His observation as to the occurrence of spreading gangrene in the flexures is one which I can also quite confirm.

The evidence upon which I rely in support of the belief that this eruption is no other than a modified varicella is the following. It occurs to children in good health at the age at which chicken-pox is common, and it affects the parts usually the sites of that eruption. It disappears spontaneously after a short time, though on account of its local severity it is always protracted longer than ordinary chicken-pox. The eruption in most instances comes out in a single crop, all the vesicles or sores being at the same time in the same stage. In the early stage it usually closely resembles chicken-pox, and is often taken for it by the child's parents. The names which it has received in Ireland of "white blisters" and "eating hives" are very suggestive. Lastly, and I think conclusively, I have seen it in case after case in which one child in a family had this gangrenous eruption, whilst the brothers and sisters had chicken-pox in the ordinary form. Dr. John Abercrombie, of the Children's Hospital, in February, 1880, brought before the Pathological Society the dead body of a child which offered a good example of the malady, and mentioned that after the child's admission at the hospital one of its brothers developed ordinary varicella.

The constitutional disturbance which attends this form of eruption frequently runs high. Dr. Whitley Stokes spoke of the disease as being often fatal, and I have myself known of several deaths. The cure, however, when healing commences, is usually rapid; all the sores, as a rule, cicatrising simultaneously.

In explanation of such exceptional severity of a disease, so common and usually so insignificant, it is impossible to do more than fall back on the suggestion of individual idiosyncracy. The severe form does not happen to delicate children more frequently than to the robust, indeed, in

several instances in which three or four brothers and sisters had varicella at the same time, whilst in only one did the eruption become gangrenous, the subject of this latter form was by no means in more feeble health than the others. I have never seen more than one child in the same family affected by the gangrenous form.

In conclusion, I should attempt a summary of the statements which I wish to offer for the consideration of this Society.

I have tried to prove that it is possible for the eruption of varicella in isolated cases and in connection with idiosyncracy on the part of the patient, to assume a very severe type, becoming bullous, petechial, or even gangrenous. In these gangrenous forms there is much constitutional disturbance, and death may result. Now and then very dangerous forms of iritis or of panophthalmitis are witnessed. Next to the assertion that there is such a malady as gangrenous varicella and in part based upon it, comes a second proposition to the effect that the *vaccinia* eruption may also assume the same type and with similar danger to life. That there does occasionally occur after vaccination a general eruption all observers admit. I have adduced only two examples of the gangrenous form of this eruption.

Respecting my own case I do not think that there can be any reasonable doubt. The child was in excellent health, was successfully vaccinated, and was affected seven days afterwards by a general eruption which was taken for small-pox. This eruption became gangrenous, and the child died of exhaustion on the twenty-first day. The eruption may have been variola, varicella, or vaccinia; that it was one of these three is almost certain. In Mr. Stokes' case the facts are similar, with the exception that it is impossible to assign the period of incubation correctly. That the vaccination sores themselves remained in both cases free from gangrene has been alleged by some critics of Mr. Stokes' case as a reason for doubting whether there was any connection between them and the eruption. But

such doubt is, I submit, not reasonable. It is the exanthem, and not the site of inoculation, which is attacked by gangrene, and surely there is no *à priori* reason for expecting that the latter should suffer.

In syphilis a very severe form of rupial eruption may follow in cases in which the original chancre was a mild one. In my own case the death of the patient deprived us of an opportunity of observing the healing of the sores, but it is to be noted that they had all at the same time advanced to pretty much the same stage. In Mr. Stokes' case the healing of the sores simultaneously gave support to the belief that they were of the nature of an exanthem, and not in any sense accidental results.

Appendix.

The following are descriptions of some cases not mentioned in the paper and of drawings which were exhibited at the meeting.

1. A portrait lent me by Dr. Barlow, from the collection in the Children's Hospital. The infant died with an eruption consisting of gangrenous patches exactly like those shown in the previous and following portraits. No history as to varicella has been preserved.

2. A portrait lent me by Mr. Waren Tay, showing the character of the eruption in the chest of a young child. Two round sores are seen, each with a central eschar of gangrene, as if punched out. In the first instance the child presented an eruption of varicella, which was seen and diagnosed by Dr. Sansom, of the North Eastern Hospital for Children. A considerable number of the spots subsequently became gangrenous, and passed into the stage shown in the portrait. Mr. Tay was kind enough to give me an opportunity of seeing the child just before the portrait was taken. I am not able to state with positiveness any facts as to family history, but I believe that varicella had occurred in the family.

3. A portrait lent me by Dr. Barlow from the collection in the Children's Hospital, showing varicella as an ulcerating, bullous eruption, not absolutely gangrenous. This portrait is of great interest as illustrating a lesser degree of severity in the inflammatory process. It shows well how large irregular sores have been formed by the coalescence of groups of bullæ. The shapes of these sores are exactly like those seen in gangrenous cases. The severity of the eruption on the trunk and head, and the comparative exemption of the extremities, is also well seen. This probably well illustrates the eruption described by Trousseau as pemphigoid varicella.

4. Portrait of an infant aged eight months, named Sawer, who came under my own observation in August, 1880, in the condition shown in the sketch. There was a history of gangrenous varicella six weeks previously. The portrait was taken in order to show the kind of scars which are left by this eruption, and to show that they might easily be mistaken for those of syphilitic disease.

The following are some particulars respecting other cases not mentioned in my paper.

Dr. David Lees gives me the particulars of a case of the child, George Watson, aged fourteen months, who was under his care as an out-patient in January, 1880. Varicella had appeared twelve days before, and four other children in the same house had it. In this child the spots had developed into deep sores, and in some on the parietal region there were black eschars appearing. One on the left cheek was so deep as almost to involve the mucous membrane. A subcutaneous abscess was formed in the neck.

Dr. Lees also gives me the notes of a child aged a year and a half, named Sarah Ann Reed, who in December, 1879, had a vesicular eruption, from which resulted a number of punched-out sores on the lower part of the back.

Also a third case, in which a female child, aged nine months, under Dr. Dickinson's care, had undoubted

varicella, had subsequently deep ulcers on the upper and inner parts of the thighs. There were no actual sloughs, but foul ulcers as large as sixpences resulted.

DESCRIPTION OF PLATE I

Gangrena lecythina in connection with *Lecythus* and *Tremella sinuata* Fuckel, p. 235.

A portion of the rock of the still close dry was shown to the Society at November 1st and a fresh eruption resembling various appeared on the 4th day after recreation. The framing was made after death which had occurred from exhaustion on the 24th day. It will be seen that the recreation spots four in number are somewhat inflated and show pus cells but they are not gangrenous. The trunk and upper parts of limbs are covered with gangrenous patches.

Plate I

Med Chir. Trans Vol. LXV

E. F. Burgess del

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DESCRIPTION OF PLATE I.

(Gangrenous Eruptions in connection with Chicken-pox and Vaccination. JONATHAN HUTCHINSON, F.R.S.).

A portrait of the back of the child whose body was shown to the Society in November, 1879, and in whom eruption resembling variola appeared on the sixth day after vaccination. The drawing was taken after death, which had occurred from exhaustion on the 20th day. It will be seen that the vaccination spots, four in number, are somewhat inflamed, and show pus scabs, but they are not gangrenous. The trunk and upper parts of limbs are covered with gangrenous patches.

Plate I

Med Chir. Trans Vol. LXV

E. F. Burgess. del

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TWO CASES

OF

CONGENITAL MACROSTOMA

ACCOMPANIED BY

MALFORMATION OF THE AURICLES AND BY THE PRESENCE OF AURICULAR APPENDAGES.

BY

JOHN H. MORGAN, M.A., F.R.C.S.,

ASSISTANT SURGEON TO CHARING CROSS HOSPITAL AND TO THE HOSPITAL FOR SICK CHILDREN.

Received October 25th—Read November 8th, 1881.

ALTHOUGH I have been unable to find any description of exactly parallel cases to those which are here detailed, they will be found to bear out the observations of previous writers on cases of a somewhat similar character, and to add to our knowledge of the appearances which may result from vestiges of rudimentary portions of the fœtal branchial apparatus.

CASE 1.—Maud Bailey, æt. 13 months, was brought to me when two months old. Her parents had been married seven years, and had had three healthy children, all of whom are alive, and free from deformity of any sort. The strictest inquiry fails to elicit an account of abnormality in any member of the families on either side. The child was very small when born, and for some weeks

did not thrive, but has made fair progress up to the present time, though it is still far from being a well-grown baby. No history of any value can be obtained as to any accident or personal injury occurring to the mother during the earlier period of her pregnancy.

The deformity was obvious, and the following peculiarities were observed when the child was first brought to the Children's Hospital.

The formation of the mouth upon the right side was natural, but upon the left its aperture extended into the cheek in a direction backwards and slightly downwards towards the angle of the jaw. The fissure by which it was prolonged was fully three quarters of an inch long, and involved the whole thickness of the cheek. It was covered with mucous membrane similar to that of the lips, and continuous with the skin of the cheek. The spot where the lips should have been united was indicated by a small papilla on either side of them, but owing probably to the absence of the entire orbicularis muscle this spot was further from the middle line on the deformed than on the natural side. Within the mouth the gum of the lower jaw was more prominent than upon the opposite side, whilst from beneath the tongue there protruded a mass of vascular mucous membrane, having much the appearance of a ranula, and which was more pronounced upon the left than upon the right side.

In the left external ear it was observed that the space between the tragus and antitragus was carried downwards towards the angle of the oral fissure and lying upon the surface of the cheek, and placed in this line were two small oval growths, one attached by a single, and one by a double pedicle covered with skin, resembling that of the tragus, in which there are small, fine hairs. No cartilage could be felt. The external auditory meatus was situated in front of the normal position, and running slightly backwards, became narrowed at its extremity, which was filled with the usual secretion of cerumen. Downwards and forwards from this ran another passage,

which terminated in a *cul de sac*, and was about half an inch in length. The lower maxilla appeared to be considerably smaller than it should have been, but seemed to be bony throughout. The movements of the lower jaw were not symmetrical.

On the right side of the face was a small growth similar to those on the left cheek, and situated in front of the tragus. The condition of the meatus resembled in a less marked degree that described as existing on the opposite side.

During the last twelve months the child has increased in size, and the condition mentioned remains the same, although the size of the fissure appears still more exaggerated.

So far as can be ascertained hearing is equally good upon both sides, and examination of the meatus (although unsatisfactory from its narrowness) does not reveal any further abnormality.

Case 2.—Elizabeth Monro, æt. 5. The parents are Scotch, and are healthy people. They have one older child, a boy, aged 9, who is weakly, but perfectly well formed. Two children were born between him and the subject of the present notice; one, a boy, died at birth, the other, a girl, died at twelve months old of whooping-cough. No deformity was noticed in either of them, nor can it be ascertained that any has existed in members of the families of either parent.

This child was very delicate at birth, and is said to have had the valve of the heart unclosed, and for some time to have been in a very critical state. She has since suffered from abscesses, which have made her delicate.

The left side of the mouth is natural, but the margin of the right extends into the cheek for about three quarters of an inch, and the edges of this fissure are covered with mucous membrane similar to that of the lips, and continuous with the skin of the cheek. Two small papillary elevations exist at the spot on each lip

where they should be united, and these are made more or less evident by muscular action, as though marking the insertion of fibres of the orbicularis muscle. There is a faint line, as of a slight cicatrix, which runs from the external angle of the fissure over the surface of the cheek towards the external meatus. No trace of this is seen on the mucous membrane, which differs only from that of the opposite side by being rather more anterior at the part which unites the upper and lower jaws. There does not appear to be any difference between the two sides of the inferior maxillæ, and although the whole bone is somewhat smaller than usual, the number of teeth upon both sides is equal. In both ears the external auditory meatus at its outer part is broad, and the space between the tragus and antitragus is wide and prolonged downwards. No marked abnormality is evident in the rest of the channel or the ossicula so far as can be observed.

Two elongated oval appendages, covered by hairy skin, are placed in a line running from the external meatus towards the angle of the fissure on the right cheek, to which they are attached by a pedunculated base, and two somewhat larger but similar growths are seen lying in the same relation on the left. The child hears equally well with both ears, although not quite perfectly with either, and she talks naturally.

Comparing the two cases, it is to be noticed that although elongation of the mouth is on different sides it is accompanied by very similar phenomena. Both cases occur in girls, which confirms the observation of Heusinger that deformities in connection with the branchial apparatus are more frequent in females. In neither case is there the history of any occurrence during pregnancy which would account for any arrest or even deviation of development; but contrary to what has been observed in cases of branchial fistula, there is no history of any hereditary tendency to aberration. The unclosed portion of the mouth extends in each case to the margin of the bucci-

nator, and although in both children this abnormal condition exists upon one side only, it is in each case accompanied by a deviation from the normal condition of both ears. While, also, the aberrant formation of the mouth points rather to deficiency, that of the ears and their appendages show, on the other hand, a tendency towards excess of growth. This alone sets at rest any suggestion as to the possibility of the deformity being due to any intra-uterine pressure or ulceration.

Very few congenital deficiencies of the body occur unilaterally, the majority being either symmetrical or in the median line. This is more especially so in the face, and the 'Teratological Catalogue' in the College of Surgeons' Museum does not contain a single specimen of asymmetrical deformity, whilst but few are recorded in the literature of this subject.

Looking to this fact, I think that the origin of the deformity of the mouth must be sought for, not in an error of formation of the mouth itself, which is developed by depression of the outer surface of the embryo above the first branchial arch, but to a failure of union between that upper part of the first visceral arch from which is developed the superior maxilla and that lower part of the same arch from which is developed Meckel's cartilage, upon which the lower jaw is formed. And this is more likely to be the case from the ill-developed condition—very marked in the one case—of the inferior maxilla, as well as by the presence and position of the "auricular appendages." Similar appendages, however, are not mentioned as existing in the only similar cases of elongation of the mouth. One of these is described by Sir W. Fergusson, where the fissure seems to have closely resembled that described in these two cases; and the second is figured in Von Ammon's 'Atlas,' and the plate is copied by Vrolik, the fissure being represented as extending upwards on the right side nearly to the angle of the eye. These are the only two cases in which this deformity has been noted.

Of the "auricular appendages," the origin suggested by Sir James Paget in his article on "Branchial Fistulæ in the External Ears,[1]" is no doubt the correct one, namely, that they are aberrant remnants of the opercular skin-fold of the first post-oral branchial cleft from which the natural auricle is developed. This opinion was coincided in by Professor Parker, to whom I showed the second of these cases. They are doubtless identical with similar growths which have been noticed by many authors as associated with branchial fistulæ, although there is no recorded case in which they have been observed in similar positions, and are said by Heusinger to be much more frequent in all domestic animals than in man. The same author states that the skin metamorphoses, as remains of the closure of the cleavings, are not unfrequently met with in the form of cicatrix-like striæ, small pore-like openings, folds of skin, and small warty-looking pieces of skin before the ears and on the throat.

Dr. Urbantschitsch, who met with twelve cases of fistulæ in 2000 patients, did not, however, once come across one of these growths. In the only case of branchial fistula which I have observed, and which occurred in a boy, and was situated in front of the right sterno-mastoid, there were no such excresences. But I have seen one case of a girl in which they existed in front of both ears as small pendulous growths in front of the tragus; and in a case of a boy in whom the right external meatus was closed and the whole auricle small and deformed, there existed a small appendage in front of the tragus of the left ear, which was otherwise naturally developed.

The cases mentioned by Dr. Allen Thomson bear a much closer analogy to the two just described, since in one of them a deformity of the ear in a young woman "was accompanied by considerable imperfection in the shape of the lower part of the face; the lower jaw was remarkably short, its position oblique, and its angle very

[1] I have to acknowledge my obligations for references both to the article, and to Sir James Paget himself, who saw these cases with me.

obtuse. The malar bone was placed far back on the cheek, and the zygomatic arch much shorter than usual. In this instance two sisters showed deficient development of the ear.

Authors Quoted.

Sir James Paget, "Branchial Fistulæ in the External Ears" (Med. Chir. Trans., vol. lxi, pp. 41—50); Sir William Fergusson (Progress of Anatomy and Surgery, p. 56); Dr. Allen Thomson (Edin. Monthly Journal of Med. Science, vol. vii, April, 1874); Dr. Heusinger (Deutsche Zeitschrift für thierische Medicin und vergleichende Pathologie, B. ii); Dr. Urbantschitsch (Edinburgh Med. Journal, Feb., 1878, p. 691), Von Ammon's Atlas, pl. IV.

Appendix.

Since reading the above paper, I have been informed of the death of the child whose condition was first described. This occurred from an attack of bronchitis, and I was unfortunately not able to obtain a post-mortem examination.

I have operated on the second child for the closure of that portion of the mouth which was abnormally prolonged and for the removal of the auricular appendages. The latter were dissected off and the former was closed by a plastic operation with a most satisfactory result, it being at the present time difficult to detect that any deformity had existed, and it is interesting to note that the action of the orbicularis has so far improved that but little difference is discernible on the two sides of the mouth when that muscle is in action.

I have since seen one other case, which, as regards the deformity, resembled those described above in every particular, the prolongation of the mouth being on the left side, but remarkable on account of its occurring in a boy, the second child of healthy parents, whose first child is a

well-formed girl, and who can speak accurately as to the absence of any similar deformity in any member of either family for three generations. The photograph of another boy presenting an almost identical deformity to that occurring in the second case described above has been kindly sent to me by my friend, Mr. F. A. Southam, of Manchester. In this case also there was no family history of abnormality.

DESCRIPTION OF PLATE II.

Congenital Macrostoma with Malformation of Auricles and with Auricular Appendages (JOHN H. MORGAN M.A., F.R.C.S.).

CASE 2.—Eliza M., aged 5 years.

Fig. 1.—Congenital macrostoma and auricular appendages of right ear.

Fig. 2.—Auricular appendages of left ear.

Fig. 2.

Fig. 1.

A SUCCESSFUL CASE

OF

SIMULTANEOUS LIGATURE OF THE CAROTID AND SUBCLAVIAN ARTERIES

FOR

INNOMINATE ANEURISM.

BY

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(COMMUNICATED BY LAWSON TAIT, F.R.C.S.)

Received October 22nd—Read November 8th, 1881.

History.[1]—John Astbury, æt. 32, puddler, was admitted into the West Bromwich Hospital, on June 29th, 1881. Has had no previous illness of note. Has no distinct history of syphilis, as although he has had sores on the penis, there have been no eruptions or sore throat since. Has four children all healthy. Fifteen years ago an iron bar, when passing through the rolls red hot, curled up and ran through his neck in front of the sterno-mastoids. Large scars still mark this injury, which on the right side extends from the thyroid cartilage to the clavicle. Has not been in-

[1] The history of this case, and most careful daily notes of it, were taken by Mr. S. A. Welch, the house surgeon.

temperate. About Christmas, 1880, had severe earache on the right side and jumping pain in the right shoulder. Continual pain between the shoulder-blades. Constant cough. Left his work early in January, 1881, the pains then being so severe that he could sleep very little, and was always worse when moving. Got very thin and ill. Could not swallow solid food without washing it down. These symptoms increased week by week until his admission on June 29th, 1881.

Condition on admission.—Very anæmic. Right pupil contracted, left normal. Free from atheroma. Continual cough. A large pulsating swelling, three inches in diameter, behind and above the right sterno-clavicular articulation, behind the sternal end of the clavicle, and bulging through the first intercostal space. Normal percussion note over the chest except over the area of pulsation, which is dull. Loud bruit over the swelling conveyed along the right carotid and towards the right shoulder, distinct from aortic heart sounds, which are normal. Right radial pulse slightly retarded, and the tracing by the sphygmograph shows increased tension. The skin over the aneurism was irritated and the sac seemed to have very thin walls and was very tender and painful.

The man took fifteen grains of iodide of potash three times a day, and was put on liberal diet of chops, eggs, and milk (he had been half starved for three months), but neither diet, rest nor drugs caused any improvement in his condition. The sac was dilating very rapidly, and both soft and hard parts in front of it seemed to be absorbing under pressure, The advisability of a speedy operation, if any, being evident, it was resolved upon consultation to apply the distal ligature simultaneously to the subclavian and carotid arteries.

On the 11th July, 1881, under ether, an incision three inches long was made through the skin and superficial fascia, which were previously well drawn down, along the anterior border of the clavicle. The skin being then drawn up, with a little dissection the omohyoid

and outer border of the scalenus were easily found, the deep cervical fascia divided and the artery exposed. The ligature was passed from below round the third part of the subclavian and tied fast. Another incision three inches long was made along the anterior border of the sterno-mastoid muscle, dividing skin and superficial fascia. Owing to the adhesions and displacements caused by the old burn considerable difficulty was experienced in finding the sheath of the common carotid artery; but by dissecting through some muscular tissue, guided by the pulsation in the vessel, the sheath was found; and, being opened, the artery was cleared and a ligature passed round it and made fast above the point where the omohyoid should have crossed the vessel. The ligatures used were of chromic catgut, which had been kindly prepared by Professor Lister, and all the details of his method were most strictly observed throughout. A bone drainage tube was inserted.

After the operation the arm was wrapped in wool and bandaged to the side. At 1.30 p.m. the same day the right cheek and arm were cold; there was no pulsation in the radial or temporal arteries, and less pulsation in the tumour. At night he had much headache. Temperature 99·4°, pulse 92. Wounds redressed on account of free oozing. The highest recorded temperature was 100·6°; this was in the evening after the operation.

July 15th.—Dressings changed and two sutures removed. A very slight trace of pus to-day, and on one other day, but only round the drainage tube. Slight pulsation in the temporal artery.

18th.—Tumour pulsating much less.

20th.—A very slight pulsation in radial artery. Patient feels very well, and can swallow easily.

27th.—Remove all sutures and find wound almost healed except where the bone tube is adherent; walls of sac feel much firmer.

August 5th.—No pulsation in the tumour over the clavicle.

19th.—Wounds quite healed. They have been superficial for some days, but have been kept open by a piece of drainage tube, which was adherent and undissolved. Tumour feels much more solid and safe. The patient is free from pain and very well.

September 6th.—The patient is up for four hours daily.

28th.—He walks about. Has no cough. The tumour seems firm. There is no bruit.

October 13th.—Patient went to Birmingham and was present at the meeting of the branch of the British Medical Association, and afterwards left the hospital.

November 8th, 1881.—Favorable conditions still continue. These may be due to the possible traumatic origin of the aneurism, as the hot iron certainly may have injured the vessel affected.

Note, June 25th, 1882.—The man has passed the winter well and has been out of doors all the time and free from cough. He has frequently pains in the right arm, and finds that he cannot lift heavy weights with it. His general health is very good. The tumour has sunk down into the chest behind the clavicle; it is smaller and seems firm. There is no bruit. The tumour moves with each pulsation of the aorta and is painless. The pupils are equal. There is *no* pulsation in the right radial or brachial, and the pulsation is so slight in the temporal as to be often doubtful whether it exists. So far the patient's condition is one of improvement, as he is better and stronger than when he left the hospital, although he has lived upon very poor fare.

CASE

OF

EXCISION OF A GRAVID UTERUS

WITH

EPITHELIOMA OF THE CERVIX,

WITH REMARKS ON THE OPERATIONS OF BLUNDELL, FREUND, AND PORRO.

BY

THOMAS SPENCER WELLS, F.R C.S.,

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(Received October 25th—Read November 22nd, 1881.)

The case which I have the honour of bringing before the Society this evening is, I believe, the first in which a gravid uterus has been completely excised in Great Britain. And it may serve to direct the attention of the profession to two important operations which of late years have been frequently performed in Germany and Italy—the one, complete excision of the cancerous uterus; the other, a substitute for the Cæsarean section, or rather an addition to the Cæsarean section of amputation of the supra-vaginal portion of the uterus.

In the following case both these objects were attained.

A uterus affected with malignant disease of the cervix, and containing a fœtus about the sixth month of intra-uterine development having been removed through the divided abdominal wall.

On the 5th of October, 1881, Dr. R. Goldsworthy Tucker, of Farningham, met at my house a farmer's wife, thirty-seven years of age, pregnant, and suffering from epithelioma of the cervix uteri.

She was married in 1872, and had five children, the youngest being sixteen months old. She had nursed this child for three months. Then she became too weak to continue nursing, and began to suffer from vaginal discharge. But she became pregnant again, and aborted after about six weeks on the 1st December, 1880.

Hæmorrhage and discharge continued after this abortion until February, 1881.

She had two regular menstrual periods in March and April, and another about the middle of May. Then she thought she became pregnant again, but was doubtful, as for one day, on the 29th May, she had some bleeding.

It was uncertain, therefore, if the beginning of pregnancy should be calculated from the middle or end of May, but she was quite conscious of the movements of the fœtus at the first visit to me. Ballottement was distinct, and I heard the fœtal heart sounds. The cervix uteri was long and enlarged, the os admitting one finger easily for a full inch, and the cervical canal was surrounded by a mass of epithelioma which everted the lips of the os and projected downwards into the vagina.

Dr. Tucker told me that Dr. Playfair had seen the patient a week before, and had discussed with him the propriety of inducing premature labour or of removing the diseased cervix. It seemed to me that the disease was so distinctly limited to the cervix that, if the diseased structure were scraped away and chloride of zinc applied to the denuded surface, pregnancy might go on to the full term. And it was settled that this should be done. But, after a few days there was so much increase of dis-

charge, and the patient suffered so much more from pain and greater weakness, that Dr. Gramshaw, of Gravesend, and Dr. Graily Hewitt were consulted; and Dr. Hewitt carefully discussed with me the various objections to, and advantagés of, the different courses open to us.

The result of this consultation was the advice that I should remove the entire uterus.

The patient accordingly came to London on the 18th October.

Dr. Hewitt and I saw her together on the 19th and on the 21st I performed the operation in the presence of Dr. Hewitt, Mr. Cadge, of Norwich, and Dr. Tucker, of Farningham. Bichloride of methylene was administered by Dr. W. Webb, and I was assisted by Mr. Thornton, Mr. Meredith, and Mr. Doran.

The patient was arranged precisely as for ovariotomy under phenol spray, but an opening was made in the waterproof covering for the passage of an elastic catheter, which was kept in the bladder, and the vagina was then plugged with thymol cotton wetted with warm water containing about 1 per cent. of phenol.

The abdominal wall was then divided in the middle line for an extent of about eight inches—from two inches above to six inches below the umbilicus.

The uterus was then turned out and was about the size of a very large adult head. Four sutures were introduced to temporarily close the upper part of the opening in the abdominal wall over a large flat sponge and thus prevent escape of intestines or cooling of the abdomen by the spray. The ovaries were seen nearer the level of the fundus than I expected to find them, and it was quite easy to secure the spermatic artery, first on the left and then on the right side, by transfixing the broad ligaments below each ovary and tying with strong silk. Then, using the catheter as a guide, I began to dissect the bladder from the anterior surface of the uterus. The uterine wall was very thin like a tense cyst, and soon gave way accidentally, the bag of membranes protruding.

I punctured the membranes and several pints of liquor amnii escaped. Then I drew out the fœtus, secured and divided the funis, but did not interfere with the placenta. I then separated the attachments between uterus and vagina all round, securing by pressure-forceps all bleeding vessels as they were divided, and removed the uterus.

Then the forceps were taken off one after another, all bleeding vessels tied with carbolised silk, the vaginal plugs removed, and the opening into the vagina and the edges of the divided broad ligaments were brought together by silk sutures. The pelvis was carefully cleansed by sponging, the opening in the abdominal wall closed by silk sutures, and phenolised cotton pads, strapping, and flannel bandage applied just as after ovariotomy.

Mr. Cadge kindly gave me the following notes which he made of the time occupied by the different steps of the operation:

2.35. Began to inhale methylene.
2.41. Catheter and plugging vagina.
2.50. Incision in abdominal wall.
2·53. Uterus drawn out.
2.56. Sutures in abdominal wall dividing broad ligament and vagina.
3.10. Uterus removed.
3.40. Ligature of vessels, and sutures of vagina and broad ligaments.
3.50. Suture of abdominal wall and dressing.
3.55. Patient in bed.

Thus the patient was about an hour and a quarter under the influence of the anæsthetic, but the operation from beginning the incision to closing the wound was completed within the hour.

The uterus has been preserved in the Museum of the Royal College of Surgeons, and I have been permitted to bring it to the meeting for inspection by the Fellows of the Society.

The following report on the preparation is by Mr. Alban Doran and Mr. Eve:

"Around the entire circumference of the os externum projects a very exuberant cauliflower mass, covered with a mucoid secretion, and at no point showing any signs of ulceration. The growth was most advanced on the right side, and on examining the deeper tissues, which it here covered, slight but marked induration was observed, extending into the cellular tissue on the right side of the uterus, close to the base of the broad ligament. The growth likewise extended, but to a very slight extent, beyond the inner border of the os uteri into the uterine cavity.

"On microscopical examination, the epithelium on the surface of the outgrowths was found to have proliferated to a marked extent, but not deeply; no "nests" could be discovered. In the indurated tissue to the right of the uterus, abundant infiltration of leucocytes was observed, without any trace of invasion of epithelial elements.

"The growth must be considered as of a kind midway between papilloma and epithelioma, a papillary growth slowly and surely becoming malignant yet not likely to recur if thoroughly removed, as in this case. The cause of irritation being removed, the leucocytes infiltrated in the cellular tissue will probably disappear, and there is little fear of recurrence.

"In fact, the *exuberance* of the growth and freedom from ulceration struck us as singular.

"The uterus and its appendages, when removed, weighed twenty-five ounces, exclusive of the fœtus, and measured six inches in length. The upper part of the uterus presented no abnormal appearance; anteriorly, immediately below the line of reflexion of the peritoneum on to the bladder, was a perfectly horizontal lacerated wound, about two inches in width, opening into the uterine cavity. The cut ends of the uterine artery could be seen on each side entering the uterus at its lateral and inferior part, between the anterior and posterior peritoneal coverings.

The right ovary contained a large corpus luteum of pregnancy, the left showed two corpore lutea in process of atrophy; the stroma of both was normal and free from dilated follicles.

"The fœtus weighed twenty-two ounces and a half, two and a half ounces lighter than the uterus and its appendages. It measured eleven inches, and was ill-nourished, its body covered with a fine down, its eyelids gummed together, and its nails not extending to the tips of its fingers; the cord was nine and a half inches in length.

"The conclusion would be that it was about a week over the sixth month after conception."

I am now permitted to add to the preceding account of the patient a short report of her progress after operation, and I need say little more than that it was very much like that after ovariotomy. There was more pain and sickness than in a simple case, but less than I have often seen in complicated cases.

Three small opiates were given within six hours after the operation.

Sickness was troublesome during the first week, and injections of beef-tea and port wine, with a little laudanum occasionally, were given every three hours. The highest temperature was 101·2° and the most rapid pulse 128. The urine was high coloured and scanty.

During the night between the 28th and 29th, eight days after the operation, she vomited frequently and there were several liquid motions; and Mr. Thornton, who saw her for me on the morning of the 29th, found that all the lower stitches had cut through, that the wound was open, and some coils of intestine visible and adherent. As I was in the country, Mr. Meredith administered methylene and Mr. Thornton replaced the stitches and separated the coils of intestine which adhered to the abdominal wall. The temperature rose to 102° in the afternoon, but the sickness ceased. After this there is very little to remark

upon, except that the second set of stitches cut their way through after six or seven days, and union was very slow. A small raw surface on one leg following a scald by a hot water cushion used during the operation was also very slow in healing.

For several days there was rather a free discharge of serum from the vagina, which afterwards became purulent and ceased within the third week.

When asked in what respect this confinement differed from those of her five children, she said she had always suffered from vomiting, but more this time than ever before; but that the chief difference was that she had no trouble this time with her breasts, and that the most pain was from the scald on her leg.

Twenty-eight days after the operation she was moved into another room. The pulse and temperature had been quite normal for several days before, the bowels acting regularly, and the urine passing freely. Appetite was good, there was neither pain nor sickness, and she slept well.

The case may, therefore, be accepted as a proof that a gravid cancerous uterus with both ovaries may be excised, and that the patient may survive the operation.

The question as to a return of the cancerous growth must remain for future observation; but inspection of the specimen affords ground for hope that the diseased part has been as completely removed as it often is in cases of epithelioma of the lip or anus, where many years often elapse without return.

If I were to repeat this operation I should modify the proceeding in several particulars.

1. The *position of the patient* facing the light as in ovariotomy proved inconvenient as daylight faded.

The opposite position, as adopted by Freund, is also in some respects inconvenient; and I think I should adhere to the position to which I am accustomed in ovariotomy unless I were to perform a combined abdominal and vaginal operation, when it would be convenient to separate

the thighs and flex the legs, keeping them well protected from chill. In any case a strong reflecting lamp might be of great service. Collin's lamp is too small for the purpose. Probably Faure's storage battery and an electric light may prove very serviceable. Until this is obtained a policeman's "bull's-eye" or a good carriage lamp should certainly be ready for use if daylight fails.

2. I expected much more assistance from the vaginal plug than I obtained. Although I pressed the cotton wool firmly all round the cervix, it was too soft to serve as a guide in separating the uterus from the vagina and bladder. An elastic ring pessary supported on a stem, or a slight modification of Zwancke's pessary, would probably be of much greater assistance as a guide both in the dissection and in passing the sutures.

3. A shorter incision in the abdominal wall would be sufficient if the liquor amnii were evacuated before commencing the incision through the os uteri and vagina, or, as soon as the uterus was exposed, by introducing a trocar as in ovariotomy.

I should certainly adopt one or other of these details in any case where the viability of the fœtus was unimportant. Even if important, a little care would protect it from injury.

4. After securing the spermatic arteries and upper border of the broad ligaments on either side, as I did by ligature, I do not think any further preliminary ligatures as practised by Freund and others can be necessary or useful, while they are very likely to endanger the ureters; and any bleeding may be at once arrested by the use of pressure forceps, as the separation of the uterus from the bladder and vagina is carried on. An elastic ligature might be very useful in any case where excision of the cervix was unnecessary.

5. By the aid of a catheter in the bladder there cannot be much danger of wounding its coats, provided the edge of the knife or points of scissors are turned towards the uterus. A hard pessary in the vagina would give

additional assistance, but I do not see how we can with certainty avoid the ureters. This seems to me to be the least satisfactory part of the operation. The surgeon cannot see or feel the ureters, and is always doubtful how near they may be to his incisions, ligatures, or sutures. It has been proposed to pass bougies through a urethral speculum into each ureter, but I have great doubt as to the ease or utility of such a proceeding. The fact that one or both ureters have been divided or tied by experienced operators will make the surgeon extremely careful to avoid the course of these ducts; but I fear that with all possible care a mistake may occasionally prove unavoidable.

6. In some of the most successful of these operations drainage has been carefully effected; and it may be necessary if antiseptic precautions cannot be effectually carried out. If they can be, I do not see that drainage can be more necessary than after the removal of uterine or ovarian tumours. And it appears to me much more important to close completely the opening from the vagina into the peritoneal cavity by sutures than to maintain an opening by a drainage tube.

7. In closing the opening between the peritoneal cavity and the vagina, it has been proposed to insert two rows of sutures, one uniting the peritoneum the other the vaginal mucous membrane. Further experience is wanted to determine which is the safer practice. My own feeling is that vaginal sutures are unnecessary, and might be injurious by preventing the escape of any blood or serum from the pelvic cellular tissue into the vagina. And they would lead to a combined vaginal and abdominal operation which otherwise may be well accomplished by the abdominal section alone.

8. Provided a combined abdominal and vaginal operation prove advantageous, a suture passed on either side of the cervix from the vagina into the abdomen, or *vice versâ*, before dividing the connections between vagina and uterus, might prove of great assistance in afterwards bringing

the edges of the opening together. A piece of strong silver wire passed well behind the ureters on either side up beside the cervix, and brought down again into the vagina near the middle line from the bottom of Douglas's pouch, would probably save much time in the latter part of the operation. I should use wire as less likely to be accidentally cut than silk.

Extirpation of the entire uterus, although successfully accomplished in this country more than fifty years ago by Blundell, has hitherto been almost rejected by British surgeons. Blundell's most successful case proved fatal, a year after the recovery, from cancerous stricture of the rectum. And it was generally felt that however painful to the surgeon, if not discreditable to surgery, it may be to abandon poor women suffering from uterine cancer to their miserable fate, it was better practice to be content with palliative treatment, or at most with removal of the neck of the uterus, rather than expose a patient to the great peril of entire extirpation. Indeed, until Freund's method of total extirpation was made known here in 1878 the operation can scarcely be said to have been seriously considered by us. And then it was felt that the cases where the disease had extended sufficiently to justify such a serious operation, and yet had not extended so far as to make total removal impossible and recurrence of disease almost certain, must be so very few that Freund's operation could only be advised in very exceptional cases. Experience during the last three years in Germany and Italy has certainly supported this opinion. Of ninety-four cases collected up to the end of 1880, only twenty-four recovered. Some died from shock, some from loss of blood, some from septic peritonitis. In six patients one ureter was divided. In two others both ureters. The bladder has been wounded, and in four cases it was impossible to complete the operation. Then, of the small number who recovered, recurrence of the disease was observed in almost every case, and in some within a very

short time after the operation. All this has led in Germany to a decided preference for total extirpation rather by the vagina than by the abdomen; and of forty-one cases recently collected by Olshausen the result is given as twenty-nine recoveries to twelve deaths. Here, again, there were cases where the operation was not performed on account of cancer but for prolapse and for uncontrollable bleeding, and there were three cases of incompleted operation.

A combination of the vaginal and hypogastric operations, as recommended in 1830 by Delpech, may probably prove more successful than the hypogastric operation in cases where the vaginal operation alone cannot be effected. But the evidence, so far as it can be estimated up to this time, is in favour rather of the vaginal than of the hypogastric operation.

Porro's operation is not an excision of the entire uterus. It is a supra-vaginal amputation of the uterus performed after Cæsarean section. It was not proposed by Porro with any reference to uterine cancer, but simply to save the woman from many of the dangers which follow Cæsarean section as previously practised. And when I was first consulted upon the case which is the subject of this paper, indeed, not until after I had performed the operation, was I aware that Bischoff, of Basle, had, in December, 1879, removed a uterus, the cancerous cervix of which was an absolute hindrance to delivery. The patient was forty-one years of age, mother of seven children, in the thirty-fourth week of pregnancy. The long cervical canal was surrounded by bleeding carcinomatous masses, so that Porro's operation, like Cæsarean section, would have left the diseased mass behind. Cæsarean section, birth of a living child, and Freund's operation were followed by great loss of blood. Transfusion led to temporary rally, but the patient died suddenly eleven hours after operation. At the examination of the body it was found that the left ureter had been tied.

My case, therefore, is not the first in which total extirpation of a gravid cancerous uterus has been accomplished; but, so far as I have been able to ascertain, it is the first which has been followed by the recovery of the patient. It is extremely improbable that operations under similar conditions can frequently become the subject even of consultation; but total extirpation of a cancerous uterus, when pregnancy does not complicate the case, will doubtless have to be seriously considered hereafter very frequently. In some cases caustics, or the cautery, or amputation of the cervix, or scraping away of the diseased growth, or a combination of these methods, may be preferred to total extirpation. But cases will certainly present themselves where the more severe operation may prove to be the safer, and where it may become the painful duty of the surgeon to afford a dying woman such hope as may be alone gained by an almost desperate operation.

Note added 21st November, 1881.—I have received to-day a letter from Professor Billroth, of Vienna, dated 18th November, 1881, which I quote, as it shows that a gravid carcinomatous uterus has been extirpated by the vagina, successfully so far as regards the recovery of the patient, but is far from encouraging as to relapse or recurrence of the disease after recovery. The Professor writes as follows:

"Your Porro-Freund case has interested me very much, as a similar case occurred to me three months ago. A strong woman, about thirty-seven years of age, four months pregnant, had extensive carcinoma of the whole cervix and part of the vagina. The whole uterus was extirpated per vaginam. Bleeding was considerable but recovery rapid. Unfortunately it was necessary to cut away part of the bladder, leaving a hole in the bladder and a gigantic hole (*Riesenloch*) in the peritoneum. I stopped up both with plugs of iodoform gauze. These were left for eight days and were then removed. There was no sepsis but recovery. The vesical fistula remains

for treatment. In another case, similar except that the uterus was not gravid, one ureter was wounded. The large peritoneal opening was plugged with iodoform gauze and the patient recovered. But I cannot heal the ureter fistula. Still, the disinfecting power of iodoform is by these cases clearly established. By no other means could the decomposition of the wound secretions, and of the urine flowing through the fistula, have been prevented, and death would have been certain.

"Unfortunately, my very successful results of total extirpation of the carcinomatous uterus per vaginam are very disappointing as to recurrence of disease. Even in the two cases just described, where I extirpated up to the extreme limits of anatomical possibility, there is already recurrence. '*Was nutzt da all' unsre Mühe und Kunst.*' Of what use are all our pains and art? cries our German friend and teacher. May we not hopefully reply that, if not invoked too late, success may reward our pains and our art."

A CASE OF LITHOTOMY,

WHERE A

TUMOUR OF THE PROSTATE WAS SUCCESSFULLY ENUCLEATED;

WITH REMARKS ON THE REMOVAL OF SUCH GROWTHS.

BY

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(Received November 19th—Read December 13th, 1881.)

I DESIRE to record this case, not as presenting any novelty in treatment or in operative procedure, but as bearing upon the remedying of prostatic enlargement by means other than those commonly recognised.

If the patient, whose case I am about to relate, had not had a stone in his bladder, he would not in all probability have been cured of his large prostate, or if his stone had been of a size or of a nature which would have permitted of its being removed by any other than a cutting operation, though he might have been relieved of it, he would have been none the more rid of those other urinary troubles which usually attend a large prostate.

To the two-fold accident of having a stone in his

bladder, and of having it of a size and constitution which did not admit of lithotrity, this patient owes a completeness in recovery which could not otherwise have been attained. Cases such as these, of which mine is not a solitary example, are instructive as suggesting whether it is not possible to apply that portion of their success which is the result of accident, rather than of design, to similar though uncomplicated conditions, where palliative measures, directed towards relieving certain consequences of prostatic hypertrophy, are found to be insufficient. It was remarked by the late Sir William Fergusson, in a communication on this subject to which I shall again refer, "When stone is combined with enlarged prostate who can say from which cause the distress is greatest?" Cases are not infrequent where the symptoms produced by a large prostate are even more urgent than those attending a stone in the bladder.

The following are the particulars of the case I desire, in the first instance, to bring under the notice of the Society:

W. B. H—, a Custom-House officer, æt. 67, was admitted into the Liverpool Royal Infirmary under my care on September 2nd, 1881, having been referred to me by Dr. Samuels. He had been suffering from symptoms of stone for seven years. I sounded him and made out that his bladder contained a large oxalate of lime calculus; further, that his prostate was enlarged.

On September 5th I performed lateral lithotomy; on seizing the stone I found it was so large that I made a bilateral section of the prostate, a course I was prepared for, and which I have successfully adopted on two previous occasions where I had to remove hard stones, weighing over two ounces.

Though this extension of my deep incision gave me additional room for extraction, I recognised that the enlarged prostate still remained an obstacle.

As a portion of the prostate seemed loose and disposed to come away, I enucleated it with my forefinger, and

slipped it out, when I was able to remove the stone from the bladder without force. There was no bleeding worth mentioning either at the time of or after the operation. The patient made a good recovery and left the Infirmary on November 5th. There was nothing to remark about the temperature during the whole of the treatment.

The tumour removed was about the size of a walnut, and it will be seen that it was almost divided into two portions by the incision into the prostate. In structure it may be described as an adenoma, analogous to what is commonly observed in the breast, and corresponding with the description given of these growths by Sir William Fergusson.

The stone was an unusually large specimen of oxalate of lime, covered with small spines, some of which were broken off by the forceps during extraction. It weighs two ounces and five drachms. By the kindness of my colleague, Mr. Bickersteth, I am enabled to give the particulars of an unpublished case very similar in many respects to that which I have related.

A country gentleman, aged sixty-three, had suffered from symptoms of stone for fifteen years. Mr. Bickersteth, considering that it was a case for lithotomy, performed the lateral operation. Finding as he had anticipated that the stone was large, he extended the deep incision and made a bi-lateral section of the prostate; this enabled him to make out that in addition to the calculus he had a large growth connected with the prostate to deal with. After extracting the stone with the forceps he shelled out with his index finger a mass about the size of a hen's egg, which proved on examination to be an adenoma of the prostate, not a true hypertrophic growth; the stone weighed nearly two ounces and a half. The patient made a good recovery, and to the present date remains perfectly well. The operation was performed on October 21st, 1878.

In 1870 the late Sir William Fergusson drew attention to the subject of lithotomy in connection with

enlarged prostate,[1] and narrated a case where, in a patient eighty years of age, after removing the stone by lithotomy he extracted the lower part of the prostate with the finger as readily as if it had been a stone. The patient not only recovered from the operation but never showed any further signs of prostatic irritation.

The paper to which I have referred concludes with the remark: "I have thus ventured to put on record what some of my professional brethren may have hesitated to do from a fear that they may have been guilty in their operations of perpetrating some rough mechanism not in accordance with that nicety of manipulation which is thought so essential in the performance of the master handiwork in surgery—lithotomy."

Mr. Cadge[2] has reported a case where he removed during a lithotomy, in the forceps between the joint of the blades, three masses which were found to be fibrous outgrowths of the prostate. In commenting upon this case, Mr. Cadge says: "In about two months the wound was perfectly healed; there is incontinence, which may be in a great part due to the removal of the prostatic tumour, but it must be remembered that it also existed before the operation. It has happened to me twice before to remove small fibrous tumours of the prostate gland during the operation of lithotomy and apparently without harm to the patient."

More recently Dr. C. Williams, of Norwich,[3] has reported a case where he removed, accidentally, between the forceps an enlarged middle lobe of the prostate. In three weeks the patient was reported as recovered, having seldom to micturate more than once in the night.

These illustrations show that two kinds of growth have been removed from the prostate gland in the course of lithotomies, namely, (1) isolated tumours, resembling adenomata,

[1] "Observations on Lithotomy and on certain cases of Enlarged Prostate." The 'Lancet,' Jan. 1, 1870.

[2] 'Trans. Path. Soc.,' vol. xiii.

[3] 'British Medical Journal,' June 15, 1878.

the term used by Sir William Fergusson, and (2) ordinary outgrowths or hypertrophies, such as we are most familiar with, as affecting the middle lobe. From the illustrations I have given I submit we may draw three conclusions at the least.

First, that lateral cystotomy may be practised in certain cases of enlarged prostrate which are attended with symptoms producing great distress with the view of exploring and if possible of removing the growth.

Second, that in all cases of cystotomy for calculus where the prostate is found to be enlarged, that a careful search should be made with the finger, with the view of effecting the removal of the growth should such be found practicable.

Third, that in determining the selection of lithotomy or lithotrity in a case where stone in the bladder is complicated with enlargement of the prostate, regard should be had to the possibility of removing both of these causes of distress by the one operation, namely, by lithotomy.

Further, the cases I have recorded seem to indicate the mode in which these growths may best be removed when met with, either in the course of a lithotomy, or a cystotomy performed for the purpose. The presence of isolated growths in the prostate can be ascertained when the gland is opened into, by exploration with the finger; for, as Sir William Fergusson observed, "as the finger passes towards the bladder, the sensation is as if its point glided through several rounded bodies in the substance of the gland, which are but slenderly in contact with each other." Of this sensation I have been conscious in more than one lithotomy I have performed in elderly persons.

Thus discovered these growths may then be enucleated by the finger as I have already endeavoured to demonstrate.

When the growths assume a more pendulous form, as we frequently see when the third lobe is hypertrophied, though they may be detached with the finger or the lithotomy forceps, as occurred in Dr. Williams' case, I

think a more precise and possibly safer proceeding might be adopted.

I refer, after the bladder has been opened and the growth explored with the finger, to the including of the growth in some simple form of écraseur by which it could be clearly detached without risk of hæmorrhage, just as is sometimes done in the case of a uterine polypus. In referring to such a proceeding, it is with the view of making preparation for what may be necessary when undertaking the operation of lithotomy in a person who is known, or suspected, to have a large prostate, the obstructing portion of which it may be desirable to remove.

In conclusion, I would remark that in none of the cases I have brought forward was the removal of the growth complicated or rendered dangerous by hæmorrhage either at the time of operation or subsequently.

A CASE

OF

FIBROUS POLYPOUS TUMOUR OF THE BLADDER SUCCESSFULLY REMOVED.

BY

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(Received November 29th—Read December 13th, 1881.)

On July 27th, 1881, Mrs. —, was brought to me by Dr. Jeaffreson, of Wandsworth. The patient, æt. 40, had been married for some years and had borne children. She was naturally of a melancholy fretful disposition; this temperament it was supposed might induce her to describe her sufferings as being more acute than others would do.

On January 3rd, 1881, having been previously free from symptoms of urinary derangement, she was seized with rigor and diarrhœa. This was followed in a few days by bloody urine, retention, and cystitis, with urgent, almost constant desire to micturate. For two or three days the urine was mainly withdrawn by catheter. On the 5th and 6th January, the urine so removed was of deep blood colour, and, though the retention passed off, the urine retained its bloody character more or less for a month. During this acute stage of the disorder the pulse was 100 and the temperature 100°—101° F. Besides bloody dis-

coloration, sometimes clots came away in ropy alkaline mucus.

During the latter part of February and March blood was no longer constant in the urine, though decolorised clots and phosphatic concretions were passed from time to time with much pain. The general health became depressed from the irritation of the bladder and broken rest at night.

No catamenia having appeared since January pregnancy was suspected, and on April 8th the uterus was examined and the bladder was sounded. In neither viscus was any condition discovered to explain the hæmaturia and cystitis. On the following day abortion of a three month's fœtus took place. The secondaries were retained for eleven days and were then removed by hand. No bad symptoms followed the abortion; nor, contrary to hope, was there any amelioration in the sufferings of the bladder. The urine continued ropy, was passed almost hourly, and every few days portions of clot or of phosphatic grit were expelled.

On June 13th, after a day of great pain, a mass of decolorised fibrine as large as an almond and coated with phosphates was passed. This was followed by considerable relief to the pain and frequency of micturition. The urine became neutral in reaction, nearly clear, and the proportion of albumen greatly diminished. The patient went to Brighton for warm sea baths, and gathered strength sufficient to be able to walk daily with comfort. Still the urine was passed every two hours both by day and night, and contained a little pus. Thus, though much better, she certainly was not well, and on her return to London, Dr. Jeaffreson brought the patient to me for consultation. I sounded the bladder, but the sound revealed no rough grating nor any unusual condition except a tender spot behind and near the neck of the bladder. In this want of precise information, and as the patient had greatly improved since the expulsion of the large phosphatic concretion, it was agreed by Dr. Jeaffreson and

myself that the cause of the cystitis might have been voided, and that the patient should try again the warm sea baths before advising that any more searching examination should be made. This improvement continued for several weeks. Afterwards the irritation returned, but without any repetition of the bleeding, which had been obstinate at the outset of the disorder. In September the patient moved from Eastbourne to St. Leonard's, where, in the beginning of October, her sufferings being very great, she consulted Mr. Penhall. That gentleman injected morphia subcutaneously and with some benefit. But as the irritation was notwithstanding very severe, Mr. Penhall, having received from Dr. Jeaffreson a full history of the rise and duration of the symptoms, dilated the urethra under chloroform, introduced his finger into the bladder, and discovered a firm protuberance encrusted with phosphates, of narrow connection, easily raised from the floor of the bladder and apparently attached to the trigone about two inches from the internal orifice of the urethra, against which it impinged by the apex. This examination caused little bleeding either from the growth itself or from the dilated urethra. The immediate result to the patient was great relief from pain, and the bladder quickly regained the power of retaining urine, temporarily affected by the dilatation. This improvement was not long continued and the urine remained alkaline with a small amount of pus. The patient's condition otherwise was good; she ate well and was in better spirits than before.

On 28th October, after consultation between Dr. Jeaffreson, Mr. Penhall, and myself, the urethra was dilated a second time with the object of removing the growth. The finger passed into the bladder and ascertained that the tumour lay just in the position in which it had been discovered by Mr. Penhall; and with a little difficulty the noose of a wire écraseur was slipped over the tumour, tightened round the base, and the tumour removed. Very little bleeding followed the extraction, and the patient's recovery was rapid and devoid of any formidable symptom.

In two days after the operation the urine was strongly acid, though it had been almost constantly alkaline or neutral for months previously.

In seven days after the operation the bladder had considerable control of its contents, and the urine had become free of pus or albumen. In a month after the operation the patient's symptoms had completely left her and she is now in good health.[1]

When taken from the bladder the growth was about the size of a small walnut, covered with phosphatic concretions, except at its base where it was almost sessile. It was moderately firm and tolerably smooth on the surface. Separated from it by the process of removal was a small pea-like translucent part, greatly resembling an ordinary polypus of other mucous surfaces. The tumour was handed to Mr. Stanley Boyd for examination, and here follows his report.

REPORT by Mr. STANLEY BOYD, M.B., B.S. Lond., F.R.C.S., Surgical Registrar to University College Hospital.

"The tumour handed to me for examination was somewhat shrunken by immersion in strong spirit. It was a roughly spherical mass, flattened from above down, having a short pedicle attached to its under surface, near one border.

"The surface was rendered irregular by low, for the most part rounded, prominences; but each of these was smooth; there were no long papillæ. Here and there was a thin coating of phosphates. The longest diameter of growth was about three-quarters of an inch.

"The growth was firm, and on section presented the appearance of moderately dense, fibrous tissue, which has been hardened in spirit. It was bounded, for the most part, by a grey layer $\frac{1}{32}$ of an inch or less in thickness, which quite conveyed the idea that the mucous membrane was continued over the growth.

[1] Dr. Jeaffreson reports (13th July, 1882) "that the patient is to-day well in every respect; without a trace of her former symptoms."

"The microscopic structure of the tumour is difficult to describe, on account of the varying relations between cells and stroma in different parts of it.

"Beginning with the surface, most of the epithelium, which must originally have covered its surface, has been lost by ulceration or by violence. Here and there a little is found in a deep recess; and it shows all the early stages in the formation of villous processes, being first divided into processes by depressions of all depths like tubular glands; then the processes lengthen, and the subepithelial connective tissue seems to grow into it; and finally a villus of considerable length was found having commencing secondary villi on its sides. It is also worthy of note that the epithelial cells are longer and narrower than those of the bladder, approaching in form those found on ordinary villous tumours.

"The narrow grey band, on most of the surface of the growth, consists of dense fibrous tissue, containing very few nuclei capable of absorbing logwood stain, and infiltrated with fine granules of earthy salts.

"This stratum is pretty sharply bounded by a more or less dense layer of small, round cells, which here and there has quite the appearance of a granulating surface. The fibrous tissue is tolerably loose here, and its fibres are more or less vertical to the surface; but it soon becomes denser, and the interlacing of its bundles gives rise to alveoli which contain masses of round cells. In some places the structure is so like that of a scirrhus that they probably could not be distinguished. The stroma in most parts contains a good many oval nuclei.

"In each section that has been examined there are two large collections of cells, which are subdivided into smaller masses by very delicate processes of connective tissue which form a meshwork. The whole exactly resembles the rarer form of alveolar sarcoma, as represented by Billroth in his 'Pathology.'

"A number of vessels enter the pedicle, and arteries of considerable size, having the usual coats well developed,

are not rare in the body of the tumour; but towards the surface vessels of any size are infrequent. Small points of calcification are common in the fibrous tissue near the surface.

"In the pedicle just above the point of section there are a few bundles of involuntary muscular fibres.

"Had nothing been known of the history of this growth I believe there would have been much difficulty in coming to a conclusion as to its nature. As it is, it seems certain that it is not malignant but that it is probably a firm fibroma. The occurrence of the different structures above described in this one simple tumour—structures which are characteristic of very different types of growth—seems to tell strongly against the possibility of drawing any hard and fast line, based upon microscopical appearances, between these types."

I must here acknowledge my great obligation to Dr. Jeaffreson and to Mr. Penhall for their kindness in supplying me with information in drawing up the report of this case. Dr. Jeaffreson's notes, taken almost daily, have furnished many details that show the unusual course of this case in the early stages. My thanks are due also to Mr. Boyd, whose description of the tumour is more complete than any published before.

Several points of interest are connected with this case. In its clinical history it did not follow the usual course of vesical tumour. First, the remarkable febrile outset. So far as I am aware, a rigor and high temperature have not been the outset of the disorder in any other case. Next retention, often a very troublesome symptom, was in this case of short duration and was not repeated.

Hæmorrhage, again, was copious at first, and long continued, either as clot or discoloured urine, but when it once ceased did not return. Repeated hæmorrhage is the great character of vesical tumour, because the frequent forms are villous or carcinomatous, which bleed freely and repeatedly. Hence copious hæmorrhage is generally a

diagnostic sign of vesical tumour. This particular growth, being fibrous, seldom bleeds copiously and usually only when injured by a catheter or sound. In Mr. Birkett's valuable paper in the forty-first volume of our 'Transactions' the unimportance of the hæmorrhage which attends the growth of fibrous polypus is pointed out as useful in diagnosis.

The chief symptom indicative of tumour was the irritability of the bladder, at times very severe and never completely assuaged. But irritability, even when stone is excluded, is not only due to tumour. In this case the abortive pregnancy, which accompanied the earlier stages of the disease, diverted suspicion, and the very great improvement which followed the extrusion of the mass of decolorised clot and phosphates in June led to the opinion that the cause of hæmorrhage, whatever it might be, had passed away. On the return of the painful micturition in October Mr. Penhall then discovered the real cause of suffering, and to him is due the credit of diagnosing the disease.

Tumours of the bladder are divided into villous, fibrous, epithelial, and carcinomatous. They are well described by Mr. Birkett in the forty-first volume of our 'Transactions,' by Mr. Murray Humphry in the sixty-second volume, and by Mr. Gross in his 'Treatise of Diseases of the Urinary Organs,' 1876. These authors state that fibroma, of which the present case is an example, is very rarely met with, most of the recorded cases being of villous tumour. Gross could collect records of only 15 cases among patients whose ages varied from thirteen months to fifty-six years, and ranging in size from that of a pea to that of the fist. This form was observed seven times in women, in most of which, if not in all the cases, the polypus protruded from the urethra. My case was an exception in this respect, for no protrusion had taken place nor could the growth be detected by the sound and finger in the vagina.

The structure of the tumour is of interest. Mainly

composed of fibrous tissue, at one part it was beginning to form villous processes, while at another it was not distinguishable from alveolar sarcoma.

This observation supports the opinion of those who maintain that any tumour projecting from a mucous surface may become villous. The presence of sarcomatous elements indicates the possibility of the growth taking malignant habit. This possible change from benignancy to malignancy is an additional reason for early extirpation.

The list of successful removals of vesical tumour of any kind is not a large one. To Gross's table of 16 cases of operation with 10 recoveries, there have been added cases by Murray Humphry, Alexander, Bryant, Lawson Tait, Norton, Heim-Vögtlin, Davies Colley, and Marcacci, and lastly my own. Together these make 28 cases with 19 recoveries and 9 deaths. In the larger portion of the recoveries the patients were females, in whom of course the procedure for removal is much easier than in males. In Alexander's case the tumours were multiple, and dilatation of the urethra was performed twice before the patient was cured. In Marcacci's case, a male, the operation was a suprapubic one, and though the patient recovered from the operation he died eventually from septic mischief derived from destruction of the pubic symphysis from urinary infiltration.

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THREE CASES

OF

TUMOUR ARISING FROM SKIN-GLANDS IN THE DOG,

SHOWING THE CONNECTION BETWEEN

DISORDER OF THE GLANDULAR STRUCTURE AND FUNCTION AND CANCEROUS INVASION OF THE CONNECTIVE TISSUE.

BY

CHARLES CREIGHTON, M.D.

Received December 13th, 1881—Read January 10th, 1882.

I. *Anatomical characters of the skin-glands of the dog; their identity with a special layer of glandular substance in the human axilla, and with a conglobate form of gland hitherto undescribed.*

The glands in the dog's skin, in which the three tumours about to be described take origin, are generally spoken of as sweat-glands, but a brief consideration of the facts will show that that designation is applied loosely if not erroneously. According to the popular belief, dogs do not perspire through the skin; and that is one sufficient reason why the glands in question should not be called sweat-glands. It is only in the sole of the foot, according

to Leydig,[1] that the skin of the dog is provided with those glomerular coils discharging by a long duct, to which the name of sweat-gland is ordinarily applied. According to the same author, the so-called sweat-glands of the dog's skin open by a comparatively short duct into the hair-follicles at a point somewhat below the opening of the sebaceous glands, and that statement is confirmed by Stirling.[2] It will appear in the sequel that the same kind of skin-glands occur also in the comparatively hairless parts of the dog and open directly on the surface of the body. Although the skin-glands of the dog differ from the ordinary sweat-glands of man both in their great size and in the character of their epithelium and the nature of their secretion, as well as in another point to be mentioned, yet they have essentially the same glomerular or convoluted plan of tubular structure as the ordinary sweat-glands, and that seems to be the chief reason why they have been generally spoken of as a large form of sweat-gland. The tube is much wider than that of the sweat-gland, and it is coiled or convoluted not in the form of a spherical glomerulus with a long duct, but in an elongated form stretching through the corium towards the surface, the duct being proportionately shortened. They may be compared to the vesiculæ seminales in miniature. The gland in section, as shown in fig. 4 (Plate III), looks like a lobule made up of a number of wide acini lined by columnar or cubical epithelium, and these acinus-like crypts or recesses of the coiled tube have a close resemblance to the alveoli of the thyroid body. They are, however, all in free communication, and the secretion formed in the deeper recesses must pass through the more superficial on its way to the outlet.

The most conspicuous and distinguishing feature of the glands, by means of which they can always be identified with great ease, is an investment of plain muscular fibres round each crypt or acinus, presenting a ribbed appearance

[1] Leydig, 'Histologie des Menschen und der Thiere,' 1857, p. 87.

[2] W. Stirling, 'Journ. of Anat. and Physiol.' vol. x, 1876, p. 471.

as regular as that of a willow basket. These unstriped muscular fibres are arranged in parallel order, with narrow intervals between them, as in a close palisade, and they constitute a substantial basement membrane upon which the epithelial cells are directly seated. In fig. 4, the basket-work of muscular fibres is represented by thin dark lines, according to the appearance of the preparation under a low power; the dark lines are not so much the muscular fibres themselves, as the highly refracting intervals between them. The fibres are about three times as broad as the intervals which separate them and their substance is for the most part transparent. One of the most characteristic appearances is that of a regular row of projecting ends of the fibres where they have been cut across. The same palisade-like arrangement is sometimes indistinctly seen in the glomerular sweat-glands of man, and it has been asserted that the latter are likewise uniformly provided with a muscular coat. That would be a morphological point of resemblance between the two kinds of glands, in addition to the convoluted tubular plan of structure common to both; but there are none the less differences between them wide enough to justify the striking out of the larger form of skin-glands altogether from the category of sweat-glands.

It is well known that skin-glands, the same as those of the dog, are found not only in other mammalians and in some batrachians, but also in the human axilla, the areola of the breast, the groin, &c. The references made to the axillary skin-glands in recent histological works are apt to convey the impression that the glands are embedded in, or in close connection with, the corium like the ordinary glomerular sweat-glands. But their most characteristic and distinctive occurrence in the axilla is not in the substance of the skin, but as a separate and circumscribed layer, of brownish colour and lobulated surface, adhering to the under surface of the skin. This glandular layer will most probably be met with at the deepest part of the cutaneous recess, over the area where the skin adheres

most closely to the axillary fascia. In a large number of observations made in the dissecting-room, I have found the glandular structure sometimes as large as a florin and as thick, but more often reduced to a few scattered small lobules, apt to be mistaken for fat lobules, and not unfrequently wanting altogether. In a microscopic section, the tubular coils are found to be separated from one another and enclosed in a large quantity of connective tissue. Good specimens of plain muscular fibres may be obtained with great ease by teasing a minute portion of the glandular structure. These observations for the most part agree with the statement of Sappey,[1] who speaks of the skin-glands of the axilla as being collected into a circular layer under the skin, three to four centimètres in diameter and two millimètres in thickness. They are described also by Kölliker[2] as forming a continuous layer under the skin, and by Frey[3] as being crowded together in a distinct stratum.

The axillary glandular stratum in man is so variously developed and so often wanting altogether, that it may be regarded as a rudimentary organ of cutaneous secretion.

The axillary odour which characterises certain individuals of both sexes is probably associated with the presence of a well-developed stratum of the glandular substance.

This view of the axillary skin-glands in man, as a separate organ in a rudimentary or obsolescent form, is borne out by an observation which I have made for another animal. In one of the Monotremes (*Ornithorhynchus*) there is a body, of the shape and size of a bean, situated beneath the skin at the lower end of the humerus. It was described by Meckel, in his monograph on that animal,[4] as the *glandula humeralis lymphatica*, and that

[1] Sappey, 'Traité d'Anatomie descriptive,' vol. 3, p. 505.

[2] Kölliker, 'Handbuch der Gewebelehre,' 1852, p. 147.

[3] Frey, 'Handbuch der Histologie,' 1870, p. 596.

[4] J. F. Meckel, 'Ornithorhynchi paradoxi Descriptio anatomica,' Leipzig, 1826.

designation does not appear to have been changed by subsequent writers.[1] Its texture, however, is more spongy than that of a lymphatic gland, and with the help of a microscopic section, it is not difficult to make out its remarkable structure. It is made up exclusively of innumerable small crypts or acini freely communicating, their walls having the perfectly distinctive rib-like structure of close-set plain muscular fibres, and their interior being lined by polyhedric epithelial cells. The structure is shown in the accompanying drawing (fig. 1, Pl. III) made by me several years ago from a section of the gland; the palisade-like arrangement of plain muscular fibres, with epithelial cells resting directly on them, is without question the same structure which occurs in the glands of the dog's skin and in the glandular stratum of the human axilla. I did not succeed in tracing the duct of the organ, and it was probably the inability to find a duct that led Meckel to describe the body as a lymphatic gland. It has the conglobate form of a lymphatic gland, and both its size and its compactness indicate a very complex degree of coiling or convolution of the continuous tube, of which it may be said to consist. I take this opportunity of putting on record an observation (with drawing) which belongs as much to comparative anatomy as to the general subject of this communication.

According to an observation of Sappey's,[2] the large axillary skin-glands in man sometimes extend to the lateral and anterior regions of the thorax; and I have in one instance found perfect examples of them in intimate association with the breast structure. The case was that of a woman, aged thirty-eight, who had a large soft cancerous tumour removed from the outer part of the mammary region by Mr. Le Gros Clark, at St. Thomas's Hospital, in 1873. I made microscopic sections of the tumour as well as of the structure adjoining it; and in the latter I found several minute cysts, about the size of

[1] Owen, Art. "Monotremata," in Todd's 'Cyclop. of Anat. and Physiol.'

[2] Quoted by Henle, 'Handbuch der Anatomie des Menschen,' vol. 2, p. 35.

a pin's head, which proved to be dilatations of the peculiar axillary skin-glands, and were so labelled by me at the time. The walls of each cyst showed, in the most exquisite form, a considerable expanse of plain muscular fibres in close parallel order; a portion of that muscular-fibre membrane is drawn in fig. 2 (Pl. III), and the characteristic large-sized epithelium seated directly on the muscular coat is shown in fig. 3. These glands were in close proximity to ordinary lobules of the breast, but there was little difficulty in distinguishing the one kind of structure from the other. As regards the tumour from whose margin they were taken, I am still unable to decide whether it should be referred to the breast, according to the original view of it, or whether it was a tumour growing from a somewhat aberrant deposit of the axillary skin-glands, and belonging, therefore, to the same class of tumours as the three from the dog's skin which I am about to describe. In favour of the latter hypothesis is the unusually large size of the epithelial cells of the tumour, and the fact that the mammary structure, as found in the piece of tissue taken from near the tumour, was everywhere quiescent and upfolded, while there were several groups of the axillary skin-glands, besides those dilated to form the small cysts, which showed a certain amount of disordered structure.[1]

[1] An appearance very similar to that of the muscular-fibre membrane drawn in fig. 2 has lately been described and figured (C. W. Mansell Moullin, "The Membrana propria of the Mammary Gland," 'Journ. of Anat. and Physiol.,' vol. 15, p. 346, April, 1881) as the membrana propria of the acini of the breast itself. The appearance was found in a tumour of the mammary region which "consisted of nothing but fibrous tissue studded with minute cysts;" it is described as a tumour of the breast. Each cyst was "surrounded by parallel rows of long tapering cell-like bodies ranged side by side with the greatest regularity, and fitting in between each other, but not nearly touching." I do not wish to pronounce a confident opinion on preparations which I have not seen, but I am sure that such a membrana propria in the mammary acini is not to be seen in any of the numerous preparations of the breast made by myself, while an entirely different kind of supporting tissue of the mammary epithelium is described by all systematic writers on the breast.

II. *Disorder of the structure and function of the skin-glands in the dog, leading to tumour formation.*

The tumours about to be described were removed during life from three dogs at the Brown Institution in 1874-5, and were carefully prepared (in bichromate of potash and afterwards in alcohol) for microscopic examination. Tumour No. 1 is a thin oblong strip removed from the back, about 3 inches long, and half as broad, and about $\frac{3}{8}$ths of an inch thick. It had a granulation-like surface, which proved to arise from a diseased condition of the hair follicles. Tumour No. 2 is a nodular enlargement, the size of a walnut, removed from a comparatively hairless region of the skin, which was unbroken over the tumour. No. 3 is a circular fungus-like growth of scirrhous hardness, with a sloughing surface, about 3 inches in circumference, and $\frac{1}{2}$ an inch to 1 inch in thickness, removed from the back. The three tumours are grouped together on the common ground of an implication of the skin-glands, but there are important differences between them. No. 1 has the disorder of the skin-glands in its deeper parts, but it has a thin layer of independent disease on the surface, starting from the hair-follicles. No. 2 is purely and simply a tumour of the skin-glands. In No. 3 there is the important addition of cancerous infection of the connective tissue. The first two taken together will serve to illustrate the initial disorder of the secreting structure and function, and the third case will be made the occasion of showing how the additional element of cancerous infection comes in.

The preparations from tumours No. 1 and No. 2 show clearly the nature of the contents of the secreting structure, or the character of the secretion. The cross section of the glandular tube is often filled with a homogeneous mucous fluid, sometimes of a brown colour; at other times the space is occupied by a heap of uniform spherical cells of granular substance and yellowish-brown pigmentation,

and without visible nucleus; and in a few instances the cells, lying free in the centre, are more or less perfect nucleated epithelium. It may be made a question whether the mucus-like secretion is produced at the expense of successive renewals of the individual cells lining the tubule, or whether the same cells may continue to give out drops of secretion from their interior for an indefinite time. But as regards the spherical granular masses, wherever such occur within the tubules, there is no doubt that they at least are the actual epithelial cells transformed and detached. They correspond to one of the "three orders of secretion" spoken of by Goodsir,[1] and, in fact, to an order of secretion less elaborated than the mucous fluid which may be taken as the ordinary functional product of the skin-glands. That the secretory product of a gland is not always fluid, but sometimes solid or cellular, is proved not only by the facts observed by Goodsir in certain glands of the invertebrata, but also by my own observations on the periodical processes of the breast.[2] Again, the thick or semi-solid secretions such as those of the sebaceous and ceruminous glands are generally admitted to be formed out of epithelial cells which have been shed bodily into the cavities of the gland.

In tumours No. 1 and No. 2, the cast-off cells of the secreting structure, or the solid products of the secretion, are found not only in the lumen of the tubules, but also, and indeed more frequently, in the spaces of the surrounding connective tissue. Fig. 4 represents the usual appearance, in section, of the skin-glands in tumour No. 1. Not only are the intervals between the coils of the tubule packed with large epithelial cells, granular and not granular, but the same cast-off cells of the secretion are found impacted in rows in the connective tissue of the corium, and these rows or processions of cells sometimes extend to comparatively distant parts of the connective

[1] Goodsir, "Secreting Structures," 'Anatomical Memoirs,' vol. ii, p. 422.

[2] 'Contributions to the Physiology and Pathology of the Breast, &c. London, 1878.

tissue. This remarkable and not generally recognised accompaniment of secretion is one that I have already described for the breast under a variety of circumstances, both of health and disease. The occurrence of precisely the same phenomenon in connection with the secretion of the skin-glands in the dog, leads me to think that the doctrine of cellular waste products of secretion, escaping not by the ordinary outlet but into the surrounding connective-tissue spaces, is a well-founded doctrine, and one that is applicable, in disease at least, to other glands besides the breast- and the skin-glands. The peculiar circumstances of the breast, in particular the remarkable periodicity of its function, give occasion to accumulations of waste products of the secretion when there is no suggestion of anything abnormal. In the skin-glands of the dog it is obvious that there is a liability, at least, to such extra-glandular accumulations of epithelial cells or cell-products, whatever be the occasion which calls forth the liability. The most singular fact as regards the skin-glands of the dog is, that the cells collected in the spaces of the connective tissue surrounding the glands are not only of the large spherical granular and pigmented kind, which are clearly enough identical with the cast-off products of the secretion, but also cells that have the cubical or polyhedric shape and the finely granular or homogeneous protoplasm of the perfect epithelium as observed *in situ*. The occurrence of both kinds of cells in the connective-tissue spaces is shown in fig. 5, drawn from a preparation of tumour No. 1 under a higher power. The one side of the figure shows the profile view of the epithelium *in situ*, and the other side shows the face view of the same, while the connective-tissue spaces round about may be seen to contain both the perfect cubical epithelial cells with a well-defined nucleus, and also granular pigmented spherical cells of somewhat larger size and with the nucleus obscured. In the preparations from this tumour, there are absolutely no instances of lymphoid cells or leucocytes in the spaces of the connec-

tive tissue, but all the cells are of large size and epithelial character. They are identical with the secreting epithelium, either in its perfect or in its transformed condition; they are found in greatest numbers in the immediate neighbourhood of the gland-lobules, and, when they are found at some distance from the lobule, it is at the same time evident from their processional grouping and somewhat compressed shape that they have been carried thither from the glandular structures within which they were produced. None of the cells have been observed in the act of passing through the wall of the glandular tube, and, in the circumstances of the case, it is hardly to be expected that that observation ever will be made. We are, in this matter, the less dependent on direct and continuous observation of the movements of cells, inasmuch as the cells are easily identified in many cases by their pigmentation, and in general by their size and epithelial character. The same kind of cells are found both within and without the gland, and those that are outside the gland must have escaped from it.

The tumour No. 1 was a thin strip of new formation which consisted partly of a hypertrophic stratum on the surface, having its seat in the hair follicles, and partly of the entirely distinct products of glandular disturbance infiltrated into the deeper parts. It is not improbable that the disease of the hair follicles may have occurred first; and, as the skin-glands open into the hair-follicles, any hypertrophic condition of the latter would probably cause obstruction to the escape of the glandular secretion and ultimately lead to that glandular disorder and epithelial infiltration which has been specially dwelt upon in connection with tumour No. 1. In the case of tumour No. 2 the almost hairless cutaneous surface is unbroken, and the tumour is owing simply and solely to the disorder of the glandular structure and function. The appearances in this tumour may be grouped under three heads:—(1) The dilatation of the glands and the multiplication of epithelial cells on their walls; (2) the enormous accumu-

lation of cellular products of the secretion in the connective-tissue spaces of the corium; (3) the cancerous infection of the fixed connective-tissue cells of the corium. The dilatation of the glands is in some parts considerable, but never amounts to cystic formation. The epithelial lining is occasionally more than one row of cells deep, and may even be raised into papillary eminences. The deeper cells are sometimes merely nuclear bodies like leucocytes or catarrhal cells; in other cases, as in fig. 6 (Plate IV), they are the large spherical pigmented elements already described. The figure No. 6 shows a portion of the wall of a glandular tube with the large cells lying both *in situ* and also in the spaces of the connective tissue. The appearance in that figure is more complex than in fig. 5 from the former tumour. It is more like a catarrhal condition, not only from the occasional admixture of nuclear or true catarrhal cells, but also from the reticular condition of the surrounding connective tissue, a condition resembling that of chronic inflammation. It is, perhaps, not going beyond the legitimate use of terms to describe as catarrhal that state of the secreting structure in which the cellular products are not discharged from the surface, but are diverted through the basement membrane to accumulate in the spaces of the subjacent connective tissue.

The second factor in the formation of the tumour No. 2 is the enormous accumulation of epithelial products in the spaces of the corium. A great part of the bulk of the tumour consists of infiltrated cells, which are usually of the large spherical granular and pigmented kind. In some places every interfibrillar space of the corium over a wide area is packed full of such cells in linear procession. It is important to observe that the cells do not appear to multiply by division after they have been carried into the connective-tissue spaces; the increase in their number is rather to be referred to successive additions from the original seat of production, viz. the glandular tubules. If the large granular pigmented cells produce by fission any of the smaller cells that are

often to be seen in large numbers beside them, they must lose their granular substance and become reduced to a nuclear state before they begin to divide.

The third factor in the formation of tumour No. 2 is the cancerous infection of the fixed connective-tissue cells of the corium; and inasmuch as that important subject is better illustrated by the appearances in tumour No. 3, I shall include what remains to be said of tumour No. 2 in the next section.

III. *Cancerous infection of the corium and subcutaneous connective tissue, following on disorder of the structure and function of skin-glands.*

Tumour No. 3 was a circular fungus-like growth in the skin of the back, with a thin upper stratum in a state of slough. It was of scirrhous hardness, and in minute structure it consisted of a dense stroma everywhere split up into numerous narrow bands, the meshes or alveolar spaces enclosing exquisitely-formed epithelial cells of a cubical or polyhedric shape with a nucleus and finely granular or homogeneous protoplasm. The tumour was evidently a cancer, but it was at the same time not an epithelioma; and it was not clear at first sight how such a tumour could be connected with any cutaneous structure. However, a more careful search within the area of the tumour brought to light two or three clusters of skin-glands; and, by way of furnishing evidence on that important point, I have drawn in fig. 8 a group of three or four such glandular crypts exactly as they lay in the midst of a tract of the stroma of the tumour. The basket-work of plain muscular fibres can nowhere be seen more clearly than in some of these tubules, and the size and form of the epithelial cells are equally distinctive. The skin-glands have been the point of departure of tumour No. 3, as of tumours Nos. 1 and 2, but in the third tumour it is more difficult to trace its structure back to the

glandular structure from which it started. Only a few crypts or alveoli of the normal gland tubules remain, these being found wherever there are any relatively broad areas of the corium left. Everywhere there is going on a formation of epithelial cells in the interstices of the connective tissue, so that the latter is split into narrower and narrower bands, and is in the end represented by single threads or fibres separating the linear rows or alveolar groups of epithelium.

That production of epithelial cells from the fixed connective-tissue cells of the corium is the prominent feature of tumour No. 3, overshadowing everything else in it. Fig. 9 (Pl. IV) gives a panoramic view of the transformation of connective-tissue cells into epithelium. The almost hidden connective-tissue cells of the corium become, throughout the whole region, plump and granular; they then appear in the form of cubical nuclei; and ultimately they acquire a protoplasmic investment, which brings them to the semblance and size of epithelial cells. But there are still traces of the original plan of glandular structure underlying that radically new cancerous process. The formation of rows of epithelial cells in the interstices of the connective tissue in many places follows concentric lines round the original gland tubules. The epithelial new formation breaks out in ever-widening circles in the tissue surrounding the tubule, so that there results an appearance as if the tubule had been greatly dilated, and as if the dilated cavity had come to be occupied with linear columns of epithelium. That extension of the area of the original tubule is not a mechanical encroachment upon the surrounding connective tissue, but it is a transforming encroachment, whereby the cells of the connective tissue become epithelial cells, and are left lying in successive concentric rows within a wide space continuous with the original lumen of the gland. The first step in that remarkable process of cancerous invasion appears to be illustrated in some of the tubules of tumour No. 2, fig. 7 (Pl. IV) being a drawing of one of them. The drawing is

taken from one side of a tubule, which shows in the other parts of its circuit the same condition that is drawn in fig. 6, viz. an accumulation of large spherical and other cells, both in the deeper layers of the epithelium lining the tubule and also in the spaces of the connective tissue nearest to the basement membrane. That part of the circuit of the tubule drawn in fig. 7 appears to me to exemplify, when compared with fig. 6, the step or the leap from a process of merely functional disturbance to a cancerous process. The connective tissue on the other side of the basement membrane is not occupied by cast-off cellular products of the secreting structure, but by a row of its own cells which have emerged from their hidden and quiescent state, have become plump and granular, and have to some extent assumed the cubical form and the regular order of epithelial cells. These characters are to be seen in the uniform row of cells next to, or perhaps within, the basement membrane, and the deeper rows of the connective tissue are following in the same direction. That abnormal formation of epithelium from the underlying connective tissue advances around the crypt or tubule in ever widening circles until the whole stroma or supporting tissue of the gland is incorporated, as it were, into the glandular structure. The cancerous infection in tumour No. 3 is for the most part the same in plan as I have described and figured, in a former paper, for a case of scirrhus of the breast; and the language which I have applied to the latter is literally applicable to the present case: "Under ordinary circumstances the row of epithelial cells on the wall of the acinus would be separated from the subjacent tissue by a more or less obvious basement membrane; whether an actual and substantial basement membrane be present or not, nothing can be clearer than the total separation of the epithelium from the tissue next to it. But here the line of demarcation of epithelium from connective tissue is broken through. . . The breaking down of the wall of partition between epithelial and sub-epithelial tissues,

as shown in fig. 6, illustrates the real nature of the cancerous invasion in the breast. The disease has spread from the glandular tissue to the connective tissue, and no limits can now be set to its progress. . . The disease has entered on a tissue which goes all through the body. The primary disturbance of the glandular epithelium has practically ceased to be the disease; it is the infection of the other tissue that now determines the extent and the rate of progress of the malady."[1]

As I have, in the paper from which the foregoing quotation is taken, and in earlier papers on tumour-infection of the liver and of lymphatic glands,[2] discussed at length the question of cancerous infection, both of the tissues in the immediate neighbourhood of the primary disease and the metastatic infection in more distant parts and organs, I shall not occupy more space here than to state briefly my view of the relation between the primary disturbance of glandular structure and function and the cancerous infection of the neighbourhood which follows it, and in the end overshadows it. There is nothing cancerous in the glandular disorder itself, and in some cases it does not differ essentially from a catarrhal disorder. The morbid cellular products of the secretion, which are for the most part large spherical granular masses, easily identified are sometimes found, as in fig. 6 (Pl. IV), occupying the spaces underneath the epithelial lining of the tubule; but they are found in greatest abundance in the spaces of the connective tissue in the immediate neighbourhood of the tubule as well as at some distance from it. Those cast-off cells of the secretion, whether they remain on the wall of the tubule or get carried away into the connective tissue round about, are not permanent and growing elements of cancerous formation, but they are the agents of awakening the cancerous formation in the quiescent cells of the

[1] "The Infection of the Connective Tissue in Scirrhous Cancers of the Breast," 'Journ. of Anat. and Physiol.,' vol. xiv, 1879, pp. 47, 48.

[2] Report of the Medical Officer of the Privy Council, &c.,' for 1874 and 1876.

connective tissue with which they come in contact. They are often found in enormous numbers in the spaces of the connective tissue, but those heaps appear to have been formed by successive additions from the original seat of production within the gland rather than by fission among themselves. Their granular and pigmented substance sometimes disappears, and it is conceivable that the nucleus left behind may become an active and multiplying cell. The large granular cast-off epithelial cells are concerned in cancer as infecting cells; and with their infecting property their activity appears for the most part to cease. They are like a fertilising influence scattered in the connective-tissue soil; they carry in them a seminal virtue which impregnates the quiescent connective-tissue corpuscles, causing them first to return to their embryonic characters, and then to develop towards an epithelial form and grouping, according to the particular pattern of the epithelial structure in which the fertilising or spermatic cells had been produced.

One of the forms of infection consists in the breaking down of the barrier formed by the basement membrane and the inclusion within the circuit of the tubule of successive rows of connective-tissue cells, infected to become epithelial; that form predominates in tumour No. 3, and an explanation of it has been already attempted with reference to figs. 6 and 7. It is more usual, however, in cancer of the breast and of other organs to find the cancerous infection breaking out at numerous points in the connective tissue, and producing linear processions or alveolar groups of epithelial cells in the midst of that tissue. There are parts of tumour No. 2 where that form of infection appears to be going on, and those are the parts to which the large granular infecting cells have been carried. In the paper already quoted, on scirrhus of the breast, I have endeavoured to show that the linear and alveolar grouping of the cast off cells of the secretion, amounting to an infiltration in the literal sense of the word, determines the often observed linear and alveolar

pattern of cancerous "infiltration" as the term is ordinarily understood in clinical practice.

Whether or not all the cast-off glandular cells have a spermatic or infecting property, it is obvious from tumour No. 1, and from parts of tumour No. 2, that epithelial infection of the connective-tissue cells does not always follow the accumulation of epithelial products in the spaces of that tissue. It requires certain favouring or predisposing conditions of the connective tissue before infection can be kindled. There are no very obvious structural or physical differences in the cutis of the three dogs, to account for the differences in the nature of their respective tumours. According to Thiersch,[1] the predisposing cause of epithelioma of the skin is a certain senile and relaxed condition of the corium. Inasmuch as cancer in general is a disease occurring after middle life, it is conceivable that there may be a condition of the connective tissue in that period which predisposes it to epithelial infection. But it is at the same time clear that there must be other reasons for that predisposition, such as heredity or chronic irritation, which are special to the individual.

[1] Thiersch, 'Der Epithelial-Krebs, namentlich der Haut,' 1865, pp. 79, 80.

DESCRIPTION OF PLATES III, IV.

Tumours from Skin-glands in the Dog (Dr. Charles Creighton).

PLATE III.

FIG. 1.—Section of the glandula humeralis of the ornithorhynchus, showing the basket-work of plain muscular fibres in the walls of the convoluted tubules. × 150.

FIG. 2.—Stratum of plain muscular fibres forming the wall of a minute cyst in the tissue near a tumour of the pectoral region in a woman. × 300.

FIG. 3.—Another portion of the same, showing the polyhedric epithelial cells seated directly on the muscular-fibre membrane. × 300.

FIG. 4.—Section of a glandular coil of the dog's skin, from Case 1, showing the infiltration of cast-off cells of the secreting structure into the surrounding connective-tissue spaces. × 120.

FIG. 5.—From a preparation of the same case, under a higher power, showing the face view of the epithelium *in situ* in the one part, and the profile view in another, with epithelial infiltration of the surrounding tissue. × 350.

PLATE IV.

FIG. 6.—Portion of the wall of a tubule in Case 2, showing large spherical granular cells in spaces of the epithelial layer, and the same kind of cells in the spaces of the subjacent connective tissue. × 350.

FIG. 7.—Another portion of the wall of a tubule in Case 2, showing the epithelial infection of the connective-tissue cells. × 350.

FIG. 8.—Remnants of normal glandular structure in the stroma of Case 3; the plain muscular fibres mostly represented (as also in fig. 4) by the dark lines corresponding to the highly-refracting intervals between the fibres. × 120.

FIG. 9.—From tumour No. 3, showing various stages of the formation of epithelial cells from the connective-tissue cells of the corium. × 350.

Fig 1.

Fig 2

Fig 3.

Fig 4

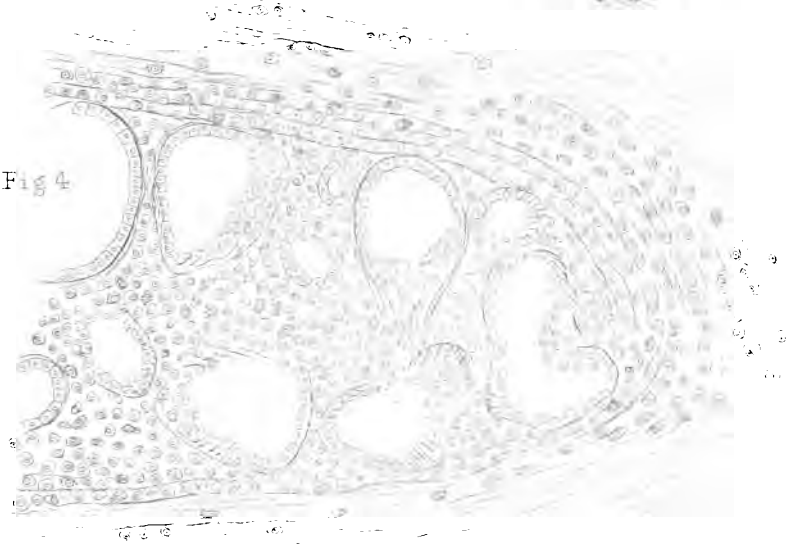

Fig 5

Plate IV.

Fig 6.

Fig 7.

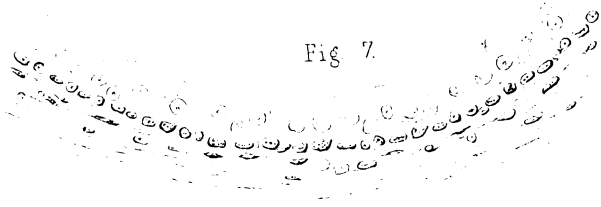

Fig 8.

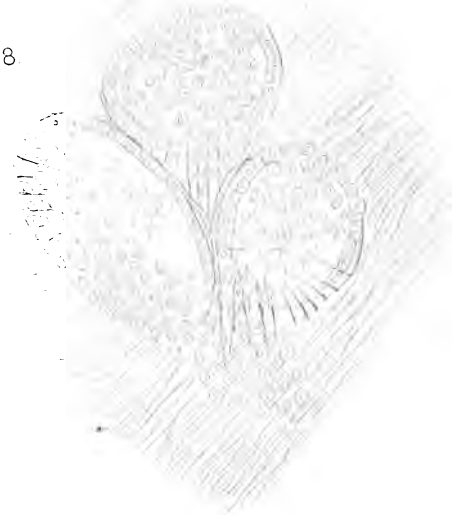

Fig 9.

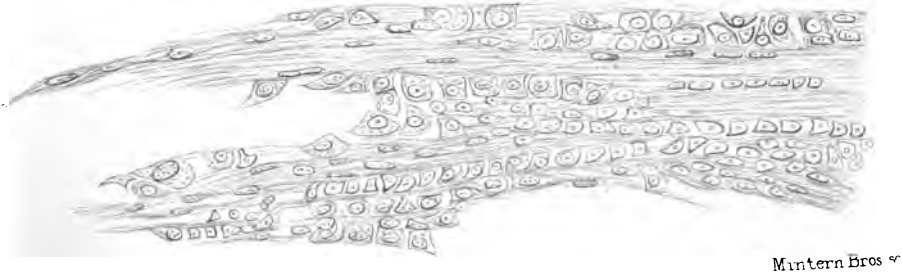

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Fig 6

Fig 7.

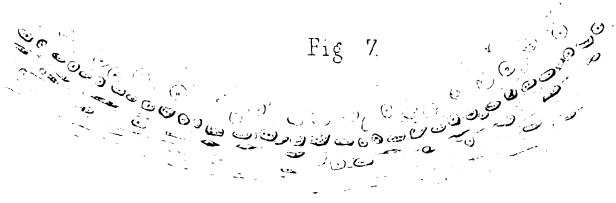


Fig 8

Fig 9

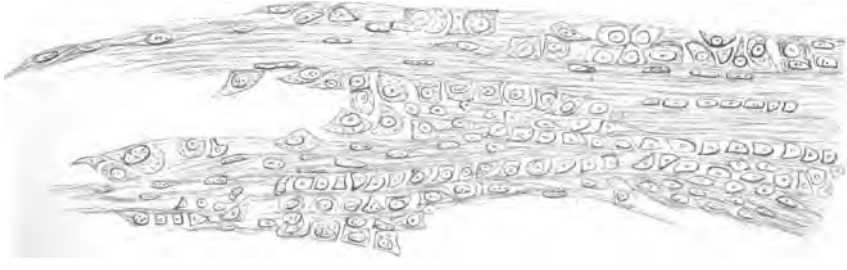


Fig 6.

Fig 7.

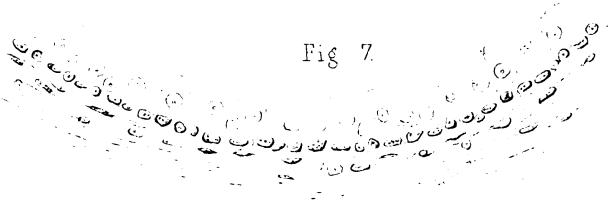
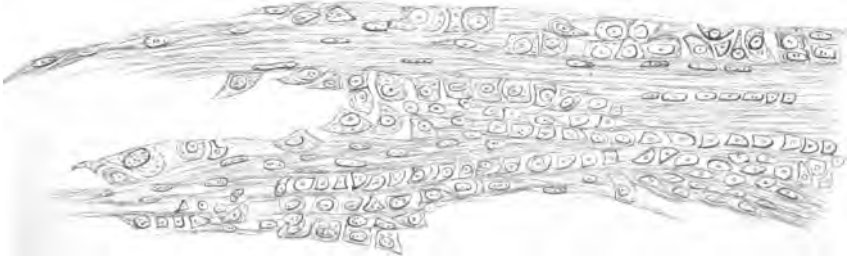


Fig 8.

Fig 9.



THE SURGICAL USES OF KANGAROO TENDONS.

BY

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COMMUNICATED BY THOMAS SMITH, F.R.C.S.

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FOR tying large vessels in their continuity the long even tendon from the tail of the kangaroo possesses decided advantages. It is as strong as the ordinary silk ligature, its knot is equally secure, and it causes no ulceration in the coats of the vessel. It has all the valuable qualities of the catgut ligature without any of the defects.

A fine tendon makes also an excellent suture, it produces no more irritation than a gut suture, and it resists the softening effects of purulent discharges for a much longer period.

In 1877 I brought the peculiar properties of these tendons under the notice of the profession in Melbourne at a meeting of the Medical Society of Victoria, and since that time they have been in frequent use there, in place of silk or gut, as ligatures and sutures. At the Melbourne Hospital they have been used on different occasions for the deligation of main arterial trunks, as the common

carotid, the femoral, &c., and they answered every requirement. The inner coats of a vessel can be divided or not, as the operator pleases, and the ends of the ligature may be cut short off. A reef-knot which has been fairly tied in a tendon will neither slip, nor will it become loose, under any circumstances. In a wound where there is little or no suppuration the tendon appears to coalesce with the living tissues. Even in suppurating wounds, where they were bathed in septic discharges, I have found the tendon sutures partly softened but still holding well together, at the end of eight days. In all the cases here referred to the tendons were previously prepared by immersion for some weeks in carbolic oil, after the manner recommended by Professor Lister for carbolising catgut. Some of our operations were performed under the antiseptic method, but others were not. *Entire* tendons only were used, those which were split being always rejected. In the hospital carbolised tendons have been also employed for making the deep stitches in ovariotomy; also for bringing together the cut edges of the vagina in the operation for the cure of vesico-vaginal fistula; in both cases they held the parts in apposition till perfect union took place. In plastic operations, in operations for the radical cure of hernia, and for the cure of varicocele, I have left carbolised tendons in the tissues, which have readily healed over them. A tendon ligature which I had applied to the carotid artery of a dog was found, ten days afterwards, when the animal was killed, perfectly strong, and the knot as secure as when it was tied, while the tendon itself was incorporated with newly formed material, and adhered firmly to the outer coat of the vessel. In this case, as one might suppose, antiseptics were not employed, and the dog tore out the sutures which held the skin edges of the wound together; therefore at the end of ten days only the deeper parts and the opening which had been made in the sheath of the vessels were healed; the rest was healing by granulation, but the ligature was not softened.

A medium sized tendon (*e.g.* about as thick as a catgut ligature which would be selected for the deligation of a large artery) is so strong that it is difficult to break it with your hands; both its strength and calibre are uniform throughout its entire length, which ranges from twelve to eighteen inches. It is owing to the peculiar flattened form, and perhaps also to the fibrous surface of the tendon from the kangaroo's tail, that a reef-knot tied in it retains its grip when left in the interior of a wound.

Like catgut, tendons can be hardened, if desired, before they are employed as sutures in certain operations, by previous immersion, for about seven hours, in a half per cent. solution of chromic acid. This preparation may sometimes be necessary, but it was not adopted in any of the cases just referred to. When hardened by chromic acid they make excellent drains for wounds, and I have often so employed them.

Their preparation for surgical purposes is most simple, but it is necessary to mention that they should not be removed from the tail *en masse*, they must be taken out one at a time, without force, or they will sometimes split longitudinally, which greatly deteriorates their strength. *A split tendon cannot be relied on, and should never be used.* In their normal state they are of various sizes; some are thick, others very fine, in fact every diameter which can be required is obtainable; there is, therefore, no occasion for splitting. Neither is it wise to twist two or more tendons together, as it destroys their flattened forms.

On removal from the recently killed animal they are cleaned first in water, afterwards in a carbolic acid solution, and then dried. They can be preserved dry, or in five per cent. carbolic oil, whichever is most convenient. It will be necessary to steep them again in a watery solution of carbolic acid before they are used.

Some tendons which I sent to London a few years ago were exhibited by the late Mr. Callender at the Clinical Society, and Mr. T. Smith of St. Bartholomew's Hospital speaks well of them. But as it has been said that they

are not at present procurable in England, I wish to add that if they are required the difficulty of obtaining supplies from Australia is almost *nil*.

THIRD COMMUNICATION

ON

ARTIFICIAL RESPIRATION IN STILL-BORN CHILDREN.

MEDIASTINAL EMPHYSEMA AND PNEUMOTHORAX IN CONNECTION WITH TRACHEOTOMY.

AN EXPERIMENTAL INQUIRY.

BY

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The occurrence of emphysema in the anterior mediastinum, and pneumothorax, in the course of a series of experiments on artificial respiration in stillborn children, has seemed a matter of sufficient interest to justify a short communication on the subject.

The series of experiments concerned twenty-six subjects who had never breathed, and extended over a period of two and a half years, from January, 1878, to July, 1880, in the laboratory of Dr. Lauder Brunton. They are related in detail in 'Med.-Chir. Trans.,' for 1881, pp. 41—101.

Five of the experiments do not concern the present branch of the inquiry, but in the others the following was the course pursued:—Tracheotomy was performed, a cannula was tied into the trachea and connected to an india-rubber tube, which in its turn was attached to a water manometer in twenty cases and to a mercury manometer in one case. When this was done, the effects of various methods of respiration by manipulation were tried, and the height of the column of water noted by the manometer.

In each case the height noted is the height above zero or the line of original level, so that the actual height of the column of water is represented by double the height indicated.

As the object was that of comparing the relative inspiratory forces of the various methods, this was quite correct as a means of comparison, though the above fact would have to be remembered in calculating the actual inspiratory force.

In the latter case the fact that the manometer would not register more than 6 or 7 inches, and had to be readjusted in the middle of the experiment when the effect exceeded this, would have to be remembered.

The accompanying table shows in the first column the number of the experiment for reference, in the second column the methods of artificial respiration employed, in the third column the maximum inspiratory effect of each method; the fourth column contains the account of the dissection and remarks.

No. of experiment.	Method employed.	Maximum respiratory effect.	Autopsy and remarks.
8	Marshall Hall Silvester (forcible) Schroeder Schultze No inflation.	0 5 in. 0 3 in.	Loud whistling heard at the root of the neck during the Schultze manipulations. Air in both pleural sacs; thymus embedded in large air-bubbles, which filled the anterior mediastinum, and on pressure escaped from the tracheotomy wound. Left lung airless. Right lung: middle lobe, and upper and inner edge of the lower lobe inflated. No wound in any of the air-passages discoverable.
11	Marshall Hall Silvester (forcible) Schultze Schultze-Silvester No inflation.	⅛ in. 11 in. 7 in. 7 in.	Gurgling and whistling heard during Schultze and Schultze-Silvester manipulations. Air in right pleural sac, none in left. Large bubbles of air in anterior mediastinum, embedding thymus, and extending along course of left phrenic nerve as far as diaphragm, and along right phrenic vein about half way to the diaphragm; on pressure air escapes at tracheotomy wound. Both lungs float, and are generally fairly expanded. No wound in any of the air-passages discoverable.
12	Marshall Hall Silvester (forcible) Schultze Schultze-Silvester No inflation.	⅛ in. 8 in. 7 in. 7 in.	Whistling heard at root of neck during Schultze manipulations. Air in left pleural sac, none in right. Anterior mediastinum full of air-bubbles, some of which escape on pressure at the tracheotomy wound. Left lung slightly inflated. Right lung better inflated, but only partially.
14 (G)	Silvester (forcible) Marshall Hall Schultze Inflation.	6 in. ⅛ in. 2 in.	Whistling heard during Schultze manipulations. No air in pleural sacs; mediastinum full of air-bubbles, embedding thymus. Both lungs fully inflated.
16 (I)	Pacini Bain Schultze Silvester (forcible) Unsuccessful attempt at inflation.	6 in. 6 in. 5 in. 6 in.	Whistling heard during manipulations (during which is not noted). Air in right pleural sac. Anterior mediastinum full of air-bubbles, which partly escape on pressure into right pleura. Both lungs partly inflated.

No. of experiment.	Method employed.	Maximum respiratory effect.	Autopsy and remarks.
18 (K)	Bain-Pacini Schüller Schultze No inflation.	5 in. 0 3¼ in.	Sucking noise heard during Schultze manipulations, the amount of inspiratory effect diminishing as this increased. Air in right pleural sac, none in left. Anterior mediastinum full of air-bubbles. Right lung airless. Left lung partly inflated.
19 (L)	Bain-Pacini Silvester (forcible) Schücking Schultze No inflation.	4 in. 5 in. 5 in. 5 in.	Sucking noise heard at root of neck during Schultze manipulations. No air in either pleural sac. Anterior mediastinum full of air-bubbles, covering right auricle of heart. Left lung airless. Right lung airless, except middle lobe and anterior internal part of lower lobe. At this point is a patch of subpleural emphysema.

Remarks.—It will be seen that mediastinal emphysema was observed in seven out of twenty-one experiments, *i.e.* in one out of three, or 33·33 per cent.

The first time the phenomena were observed, it occurred for a moment that the emphysema might be due to post-mortem changes, but the freshness of the subject contradicted this.

The next impression was that the air had escaped from one of the bronchi or the trachea, but careful examination and inflation through the trachea with the extremities of the bronchi clamped failed to discover any leak.

It was not long before the appearances could be satisfactorily traced to their cause.

Pneumothorax was often associated with mediastinal emphysema, but never occurred without emphysema; on the contrary, the emphysema occurred without pneumothorax. Therefore the pneumothorax was probably a later sequel of the emphysema.

Air was observed to escape from the mediastinum into the pleural sac.

Air could be pressed from the anterior mediastinum and emerged at the tracheotomy wound.

A whistling or sucking noise was observed during the experiments at the root of the neck.

It appeared, then, that the air travelled from the tracheotomy wound into the mediastinum, which in some cases it ruptured, producing pneumothorax.

Pneumothorax occurred in five out of seven cases of mediastinal emphysema, in one case into both pleural sacs, in three into the right, in one into the left sac.

In every case but one (16 (1), in which both lungs were about equally inflated), the pneumothorax occurred on the side on which the lung was less expanded.

Experiment.—By way of confirmation a fœtus was taken, and the tissues down to the trachea were divided, the trachea being left intact; the skin was raised so as to form a funnel or pouch, and into this coloured injection was poured. Silvester's method was then employed; the coloured injection was found in the anterior mediastinum.

The following is the explanation offered:

When by the manipulations for respiration the thoracic cavity is expanded, the intra-thoracic air is rarefied (which is relatively the same thing as the extra-thoracic pressure being increased).

When all the air apertures of the thorax are closed the only way in which the intra- and extra-thoracic pressures can be equalised is by suction through the blood-vessels; the lungs become hyperæmic, and transudation of serum into the air-passages may occur, causing œdema.

If all the air passages are not closed the pressure will be equalised through the route of least resistance—the trachea.

This is what occurs in ordinary breathing:

When a manometer is tied into the trachea, the air enters by the trachea until the column of fluid equalises the pressure, when equilibrium is established.

But the weight of a column of fluid is equivalent to an obstruction of the air passages; and in this case the

same phenomena may be observed in the subject as are observed in a living child which is making violent inspiratory efforts, its air passages being obstructed, viz. the pressure of the external air depresses the weakest points.

In a living child these include the supra-clavicular fossæ and supra-sternal notch, the abdomen, and hypochondria.

In a dead child the effect of the diaphragm (which is practically not much affected by any artificial mode of respiration) is eliminated, and the hypochondria are not retracted, but the abdomen and parts above the sternum and clavicles are depressed.

But if a potential passage to the thoracic cavity exists, this becomes a *locus minoris resistentiæ*, and the pressure will act through it.

The tracheotomy wound establishes this potential passage by piercing the deep cervical fascia. This fascia, as is well known, runs behind the sternum and becomes continuous with the sheaths of the great vessels and the pericardium.

It is through this passage that air penetrates into the mediastium.

Whether it goes further than this is determined by the question whether a sufficient quantity can be contained there to equalise the pressure; if not it will go farther.

In one case it followed the course of the phrenic nerves, on one side to the diaphragm.

In the majority of cases it burst the mediastinum and distended the pleural sac.

It was observed that in most cases one lung was better expanded than the other, and it was on the less expanded side that the pneumothorax occurred.

This is what we should have expected from the above-named physical conditions.

No case occurred in which it burst into the better expanded side, though in one case, the lungs being (as far as could be seen) about equally expanded, pneumothorax occurred on one side only. (A thickened pleura,

however, might determine rupture into the opposite or better expanded side.)

In one case (19 L) it was noted that with mediastinal emphysema there was one patch of subpleural emphysema on the solitary expanded portion of either lung.

With emphysema in connection with partial collapse, solidification, &c., we are familiar; it is but an illustration of the same physical laws as preside over mediastinal emphysema.

It was observed that the noise at the root of the neck in six cases out of the seven was found to occur during the Schultze manipulations; in the seventh case it was heard, but its exact time of occurrence is not noted.

This coincides with what we know of the suddenness and violence of the action of this method noted elsewhere; it was the sudden jerk caused by this method which produced the sudden blast at the tracheotomy wound which burrowed into the anterior mediastinum.

It is to be remarked that the presence of a large mediastinal emphysema, even with pneumothorax, does not necessarily prevent considerable inspiratory effect.

I have looked through the post-mortem records of St. Bartholomew's Hospital from 1868 and find records of twenty-seven cases of autopsies after tracheotomy for various causes.

Mediastinal emphysema is recorded in three cases, or 11·1 per cent.

In two out of these three cases pneumothorax is noted, and in one it was the cause of death. In one case in which pneumothorax was probably bilateral, both lungs were collapsed; in another, the right-sided pneumothorax corresponded with right-sided collapse.

In one case there was emphysema of the tissues about the neck. In one case the air channel was traced down from the wound into the anterior mediastinum.

I have also examined the post-mortem records of the Hospital for Sick Children since 1860, and find records of eighty-two cases of autopsies after tracheotomy for various causes.

Mediastinal emphysema is recorded in five cases, or 6·09 per cent.

In no case is pneumothorax noted.

The state of the lungs was as follows:

Case 1. Both lungs collapsed.

2. No collapse.

3. Both lungs partly hepatised.

4. Anterior parts of lungs emphysematous, posterior parts less expanded than anterior.

5. Upper lobe and posterior part of lower lobe of right lung solid and airless.

Emphysema of the neck is noted in three out of the five cases.

I do not imagine that these numbers represent the actual facts; indeed, in the cases in which mediastinal emphysema is recorded, it is almost invariably mentioned by the way as if an unimportant fact; and one recent case was only recovered from oblivion by personal inquiries.

This is not to be wondered at, considering that, as far as I have been able to discover, the matter is not even hinted at in any of the books dealing with tracheotomy; indeed, the following passage is the only mention of the subject which I have been able to find:

Wilks and Moxon, 'Lectures on Pathological Anatomy,' second edition, 1875, p. 308:

"We believe we have seen two cases of pneumothorax arise from tracheotomy, and we mention the circumstance because we are not aware that it has ever been alluded to. In one case where, after tracheotomy, death occurred without sufficient reason, both lungs were found contracted in the chest, and the cellular tissue in the posterior mediastinum was filled with air, producing large bubbles, which we think had burst through the pleura into the chest.

"In another case, where most extensive superficial emphysema followed the operation, the breathing became laborious before death, and the lungs were found con-

tracted in the same manner; the emphysema had penetrated the mediastinum."

The latter of these two cases, together with my experiments, may illustrate the production of emphysema of the tissues of the neck, &c., during labour.

With regard to pneumothorax, any small amount of air in the pleura is liable to escape observation unless the thorax is opened under water.

Still, thus much is proved:

1. That mediastinal emphysema does occur after tracheotomy.

2. That it is sometimes accompanied by pneumothorax.

3. That it sometimes exists apart from emphysema of the neck.

The route which the air takes was proved by experiments to be behind the deep cervical fascia.

It remains to discuss the relation of mediastinal emphysema to emphysema of the neck.

It would appear, *à priori*, probable that these would be due to a common cause.

The experiment in which coloured injection penetrated beneath the deep cervical fascia to the anterior mediastinum, without tracheotomy; and all the experiments in the table (in which no escape of air from the trachea was possible) prove that the air does not penetrate from the tracheal wound.

But if the above views are correct the two sorts of emphysema are obviously due, not only to different but to opposite causes, mediastinal emphysema being due to the diminished pressure in the thorax during inspiration, emphysema of the neck, on the contrary, being produced during expiration.

Emphysema of the neck may indeed run beneath the skin and fascia upwards to any extent, and downwards over the thorax, but the air cannot be forced by the expiratory force into the thorax, for the simple reason that the *vis a tergo* of the air issuing from the tracheal

wound is at once met by the equally powerful *vis a fronte* of the intra-thoracic pressure.

The production of anterior mediastinal emphysema and the route taken by the air need not be again explained.

It is not to be denied that air blown from the tracheal wound beneath the cervical fascia may be subsequently sucked into the anterior mediastinum (though air in the subcutaneous cellular tissue could hardly do so), but what is insisted on is that, whether the air travels directly from the wound or indirectly, as explained above, the mechanism of the production of the two sorts of emphysema is entirely different, and, indeed, opposite.

The following case shows that blood or, indeed, any other fluid, effused beneath the deep cervical fascia, may subsequently be drawn into the thorax, a fact which perhaps deserves attention with regard to the after treatment of operations near the root of the neck and of deep cervical abscesses. In view of the above observations, strong or sudden inspiratory efforts (*e.g.* in vomiting) must greatly increase the risk of thoracic complications from the inspiration of pus or other fluids into the thoracic cavity.

The case is briefly as follows:—Induction of premature labour for flat pelvis; turning; delay of after-coming head at brim; child stillborn, with heart beating; artificial respiration by direct inflation and the methods of Silvester and Schultze for three hours, after which child breathed naturally; death six hours after birth.

Autopsy showed separation of fifth and sixth cervical vertebræ without fracture, blood extravasated in all directions, among others into the tissues of the neck, including deep cervical fascia, from whence it extended downwards to the apices of the lungs and along the sheath of the vessels on the left side to the level of the third dorsal vertebra. Bloody fluid in both pleuræ.

It is of some consequence to consider the exact moment at which the danger of mediastinal emphysema occurs.

The conditions favouring its production are two—a

wound of the deep cervical fascia, and obstruction to the air passages; the moment of its production is inspiration.

To apply this: the danger begins as soon as the operator has divided the deep cervical fascia. The lower this is divided the more easily emphysema of the anterior mediastinum is produced.

Supposing obstruction to exist in the larynx, the period of greatest danger is the interval between the division of the deep cervical fascia and the establishment of efficient patency of the trachea.

Elevation of this fascia away from the trachea renders the entry of air beneath it more easy.

Bungling in inserting the tube increases the danger by prolonging the dangerous period.

Insertion of the tube beneath the deep cervical fascia instead of into the trachea (of which instances are on record) renders the entrance of air easy above all other conditions.

The following practical conclusions are offered:

1. Emphysema of the anterior mediastinum occurs in a certain number of tracheotomies.

2. It is often associated with pneumothorax, to which it stands in causal relation, and which may be the cause of death after tracheotomy.

3. The air is most likely to burst into that pleura of which the lung is the less expanded. On the other hand, pneumothorax, of course, helps to collapse the lung.

4. The route selected by the air is the space beneath the deep cervical fascia.

5. Emphysema of the anterior mediastinum may or may not be associated with emphysema of the neck; but their causes are different, and the conditions of their production are opposite.

6. The conditions favouring the production of mediastinal emphysema are division of the deep cervical fascia, obstruction to the air passages, and inspiratory efforts.

7. The dangerous period during tracheotomy is the

interval between the division of the deep cervical fascia and the efficient introduction of the tube.

8. The incision in the deep cervical fascia should not be longer than necessary in the direction of the sternum. It should on no account be raised from the trachea, and this should be particularly remembered during inspiratory efforts.

9. It will probably be found that the frequency of occurrence of emphysema of the anterior mediastinum depends much on the skill of the operator, especially in inserting the tube.

10. If artificial respiration should prove necessary the tissues should be kept in apposition with the trachea, and any manipulations performed steadily and without jerks.

11. Schultze's method (which is not otherwise suitable for the above purpose) is especially prone to produce emphysema of the anterior mediastinum.

12. These observations illustrate the fact that, apart from the question of tracheotomy, the inspiratory force of the thorax should be remembered in all operations near the root of the neck, whether dealing with vessels or not, and in the case of all collections of pus beneath the deep cervical fascia. In these cases quiet respiration is essential for the safety of the patient, and vomiting, which begins with a sudden inspiration, is dangerous.

13. These observations may serve to illustrate the production of emphysema of the neck, &c., during labour.

ON THE GREAT FREQUENCY

OF

CARDIAC MURMURS IN THE PUERPERAL STATE.

BY

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COMMUNICATED BY DR. JOHN WILLIAMS.

(Received January 3rd—Read February 28th, 1882.)

THIS article is the outcome of a routine examination of the hearts of lying-in women. The opportunity for its production has been given me by the kindness of Dr. John Williams and Dr. Champneys.

The observations were made in the wards of the General Lying-in Hospital, Lambeth, and lasted over a period of six months, from February to August, 1881. The number of cases received during that term was 111; these were taken consecutively, not picked or selected. The kind and amount of examination has been of this sort. The women entered actually in labour, when a a short present state was taken prior to delivery; their hearts were then investigated by the ordinary means at least once every morning during the time they were

resident in hospital, which was almost uniformly a fortnight; in addition to this, the visiting physician examined twice a week in the afternoon.

The chief result which this communication has to chronicle is the remarkable frequency with which murmurs were encountered over the præcordia of the ordinary hospital lying-in woman. I have good grounds for believing that these abnormal sounds occur outside the hospital in patients confined at their own homes.

Out of the 111 cases a murmur was heard in 84, or roughly about 75 per cent.

I have judged it best to tabulate the murmurs according to that part of the area of the præcordia over which they had their maximum intensity. Thus:

	1st sort.	*2nd sort.*	*3rd sort.*	*Total.*
Tricuspid.	36	29	=	65
Pulmonary	6	—	7 =	13
Mitral	8	—	2 =	10
Aortic	1	—	7 =	8 = 96

The murmurs are here arranged into three sorts; the apparent discrepancy between the result thus obtained and the former statement is due to the fact that cases were examined having more than one bruit.

It will be noticed that, contrary to what is met with in mixed practice, the seat of the majority of the murmurs is over what may be termed the right heart; so much is this the case that, instinctively after a time, one came to apply the stethoscope first to the left of the lower part of the sternum.

I shall now state, as summarily as possible, some facts which come out as the result of observation directed to the state of the heart's action and other features in these puerperal patients generally, before passing on to the subject proper of the bruits. An induction is made to the effect that what has been observed in these hundred and odd cases applies to all lying-in patients.

It is believed that in the puerperal state the right

heart is acting strongly. The pulmonary second sound was very commonly markedly accentuated, and it might be reduplicated and, sometimes very much changed in character, even to closely imitate, and, I believe, in a few cases actually to be a diastolic murmur. The first cardiac sound over the right ventricle also indicated robust action, and this sound was frequently reduplicated.

The colour of the conjunctivæ and mucous membranes has been recorded in every instance, and some investigations have been made with Dr. Gowers' modification of Hayem's hæmoglobinometer.

As was to be expected, a large proportion of the cases were paler than natural (67 per cent.); the average pallor in the language of the hæmoglobinometer was 70 to 85 per cent. of the normal colour standard. It has been considered that 90 per cent. is quite a metropolitan healthy standard. One multipara, living all her life in London, had cent. per cent. of hæmoglobin; this patient had a murmur of systolic time over the right ventricular, mitral, and pulmonary areas, of maximum intensity about the fourth left space, close to sternal edge; this murmur developed on the first day after delivery, and was heard for two mornings following, after which the first sound was altered in quality and slightly prolonged, but this feature disappeared, and the patient left the hospital with quite healthy sounds.

The urine was investigated daily in every case. The information thus gained has only a negative value; no accompanying change was made out between any of the characters of the urine and the occurrence of the murmurs. The urinary observations have their value in other ways.

The temperatures of the patients have been taken as systematically as possible every four hours. A fact of importance has come out in connection with this subject; it is that the bruit often had its intensity greatly augmented, and in some instances was only developed, and existed during a pyrexial period.

The occurrence of rheumatism in any of its protean aspects has been sought for in all the cases; the result is of negative value as regards the great bulk of the murmurs.

No one mark or set of marks in common seemed to exist, and there seemed to be no truly causational element in the factors of age, civil state, or number of pregnancies. We have broadly stated the numerical and positional strength of the murmurs; it is necessary now to make use of an appropriate arrangement in order to obtain further acquaintance with this vast and varied body.

Two cases of undoubtedly structural heart disease will be first separated; no further reference will be made to these. The great bulk of the murmurs remain to be dealt with. These murmurs may be divided by certain fairly well-marked characters into three kinds. Each of the three sorts will be fully treated of, both as to description and discussion of mechanism, before the next is considered, as tending to clearness of exposition.

The whole of the murmurs had one feature in common, that was their invariable systolic rhythm.

The First Sort of Murmur.

The first sort (resembling an ordinary endocardial murmur[1]) was of blowing character, soft, usually low or medium pitched, fairly long, and heard with almost every cardiac stroke, presented but little variation during the whole course of its existence. The great majority of this kind were loudest over the right ventricular region, about the fourth left space and little to the left of left sternal edge.

They were thus distributed:

[1] Phrase used as conveying in two words a description.

Seat of Maximum Intensity.		*Conductibility.*	
Right Ventricular or Tricuspid	36	Unconducted	15
		Heard all over the præcordia	7
		„ over pulmonary	9
		„ „ „ and mitral	3
		„ „ „ „ aorta	1
		„ „ mitral	1=36
Mitral	8	Unconducted	3
		Heard over tricuspid	4
		„ all over the præcordia	1=8
Pulmonary	6	Unconducted	2
		Heard over tricuspid	2
		„ „ „ and mitral	2=6
Aortic	1	Heard also over pulmonary	1
		Total number of murmurs of 1st sort	=51

This is a summary view of what may be taken to be the varying conductibility of the murmurs having their maximum intensity over one or other of the four areas.

A murmur of this class was associated five times with one of the third class, three times with one of the second class.

This murmur possessed the most constancy; it was not, however, unimpeachably so. At times it altered its seat of maximum intensity, and at times even its existence might be called in question; the extent over which it could be heard varied even from day to day; it might even disappear so that nothing more than an impaired first sound could be said to exist, though there was no telling whether it would or would not return in all its former strength. No doubt this sounds fanciful, but that epithet belongs to the murmur, not to the observer. This murmur was rarely heard before delivery. Most commonly it developed during the next day or two following labour.

A notion which almost naturally suggests itself, seeing the frequency of the murmurs during the puerperal state, is whether these murmurs have such frequent presence during the pregnant state. In relation to this a satisfactory conclusion has not been come to.

Patients obtaining letters for the institution were examined once in the later months of pregnancy, and again on admission prior to delivery.

The tendency of these observations is to show that the bruits are by no means so frequent either during gestation or during labour as they are in the lying-in condition. Examination during a second stage labour-pain is obviously difficult and unsatisfactory in most cases.

In one case a murmur was distinctly heard and noted when the patient obtained her letter a month before confinement, but no murmur was made out during or at any time after labour.

It is probably true, but this is regarded quite as a tentative statement, that these murmurs are for the most part appanages of the puerperal state.

The duration of the murmur was very variable; supposing it to appear on the first or second day of childbed, it might last for two or three days or a week, or be heard up to and on the fourteenth day—the day of discharge. Twenty were heard when the patient was about to leave the hospital.

These cases should have been seen again at the end of a month, but, unfortunately, few returned.

Three of them were carefully examined after the month; in these no murmur was detected. Four retained their murmur, and these were obviously still anæmic, notwithstanding medicine. Of those who never had a murmur during childbed, some showed themselves at the end of the month and were then murmur free.

The first sound of the heart over the area, auscultation of which subsequently detected a murmur, was often markedly altered in quality and prolonged; and when it might be inferred that the murmur was disappearing this impurity of the first sound was again noticeable, the murmur being said not to exist, and yet the first sound still having or being accompanied with markedly changed features.

It is thought probable that the conditions originating

the first sound are variable, that the murmur is the furthest expression of this variation, an implication being that the whole or part of the antecedent or antecedents of the first sound may or can vary even in a short space of time.

Discussion of Mechanism.

Having described what has been observed and inferred with respect to this first sort of murmur, I may now pass on to the consideration of their mode of production.

The basic murmurs are regarded as of "hæmic" origin, without necessarily implying finality in our knowledge of these sounds.

The chief interest, it seems to me, fixes itself on the subject of the mechanism of the apical murmurs, and more especially of those seated over the right ventricle.

We have eight mitral murmurs to consider. Four of these are called permanent, as they existed from the beginning to the end of the case whilst under investigation.

None of the mitral murmurs were audible in the back of the patient, neither in the vertebral groove, nor at the inferior angle of the scapula.

Two permanent murmurs had a distinct history of rheumatic fever with joint affection; in these the apex beat and impulse observations indicated some hypertrophy.

Walshe says: "If the murmur be inaudible posteriorly, I believe it very seldom depends on regurgitation of structural mechanism; possibly it may never do so, but I am unprepared to adopt so sweeping a statement." Further, "Systolic apex murmur inaudible posteriorly is the exponent of dynamic mitral reflux or of various conditions, unnaturally soniferous, occurring in the interior of the ventricle." The "various conditions" mentioned in a tabular form by Walshe are "roughness of endocardium of ventricle or of inferior orifice of valve; mis-

attachment of a tendinous cord; thickening and roughness of cords; fibrinous threads crossing line of current; coagula in cavity." Abnormal collision of blood is the mechanism assigned as producing the murmurs when these "various conditions" are present. The remaining two permanent bruits had no history of rheumatism. One was aged twenty-six and 2-para, the other aged thirty-six and 5-para: in both there were signs of hypertrophy. In all four the pulmonary second sound was markedly accentuated.

The four murmurs were probably dependent on some one of the "various conditions, unnaturally soniferous, occurring in the interior of the ventricle," an interesting element of this question being the possible origin (in two of the cases) of the murmur-producing mechanism during the pregnant and puerperal states. This subject is treated by Porak in his recent work. The four transitory murmurs must have had some equally transient antecedents.

The possibility of altered dynamic conditions being the *rationale* of these is favoured by recorded altered position of the apex beats in these cases whilst under observation[1] and by the known nervous instability of puerperal patients. Under this heading would come those murmurs heard in the mitral area not due to conduction, but being associated with a murmur elsewhere, yet holding a subordinate position, at least, in intensity. In one of the temporary mitral murmurs there was a history of rheumatic fever with joint affection.

The late Dr. Pearson Irvine advocated the mechanism of ventricular dilatation for murmurs occurring in chronic anæmia; this explanation, with its molecular alteration of structure of the ventricular parietes, will not hold, I think, in the above cases.

The murmurs of maximum loudness over the right

[1] Errors in observation are apt to creep in. The callipers were used and the measurement taken from mid-line of sternum to apex beat. Allowance being made, I still think the observation really holds good, as the difference was from ¼" at least to ½" at most.

ventricle are, to my mind the most interesting and the most novel.

I have been able to find only two papers which deal in any definite manner with murmurs of this situation. Parrot, in 1866, wrote an article on so-called anæmic murmurs, which he contended were produced at the tricuspid orifice, since they were loudest over the fourth left costal cartilage, there or thereabouts.

In the 'Archives de Tocologie' for March, 1881, Dr. Letulle has made out the existence of these sounds, though far less frequently, in childbed. His examinations were few, and so he missed a great number of these abnormal sounds. The murmur which Dr. Letulle described is sufficiently like the one I have endeavoured to pourtray to establish its identity. Dr. Letulle, like Parrot in the anæmic cases, regards the sound as indicating tricuspid regurgitation. He noted fulness and pulsation of jugular veins; increased transverse cardiac dulness pointing to dilatation of right heart.

I do not believe that the jugular pulsation exists apart and distinct from motion communicated by contiguous arteries. Moreover, in the presence of such vitiating and variable conditions of skin, fat, and breast, as may be met with in childbed, do I believe it possible to percuss to such niceties as Dr. Letulle evidently does.

In fact, to my mind, outside the existence of the systolic murmur, I do not think there is any sufficient evidence of tricuspid regurgitation.

That tricuspid back flow may take place, and be manifest to our senses only by a systolic murmur, or that it may occur with no outward sign, may be regarded as conceivable and perhaps possible.

Another suggestion may be made. Is it possible that these murmurs are really concerned with the outflow of blood from the right ventricle in its natural, rather than in its unnatural direction? That the murmur is direct rather than regurgitant.

The view that all these functional murmurs must be

produced at a cardiac orifice does seem to me rather unreasonable. Dr. Gowers has told me that he considers the notion that the murmur may originate in the ventricular cavity to be not at all an improbable one.

Physiology teaches us that during the progress of the systole the great arteries elongate as well as expand so as to put the base of the heart at a lower level. We also know from the same source that though this does occur, yet the apex beat remains stationary, this being due to the "gathering up" of the heart's muscular substance. The needle passed through the chest wall into the apex merely quivers, whilst that at the base moves upwards, indicating a downward dislocation. The tension in the pulmonary artery is, I believe, increased in the puerperal state, and so we might expect to find a lowering of the apex beat, continuing whilst the extra tension continued. Is it possible, therefore, that there is a migration of the pulmonary orifice downwards during the early part of childbed? Would this be sufficient to explain the fact that these systolic murmurs have their greatest intensity as low as the fourth space? My observations will not bear straining to meet such a hypothesis.

I prefer to leave the question open: the *rationale* of murmurs is altogether too puzzling to be easily settled.

The Second Kind of Murmur.

This second sort (friction-like) was systolic in time, was almost absolutely non-conducted, being heard only over a very small area to the left of the median line of the sternum, it sounded quite superficial, and was short and high-pitched and rather stiff in quality. This friction-like systolic murmur was to be heard just above and to the left of the ensiform cartilage; it was usually audible only over an area which might be covered by the small end (of moderate size) of the stethoscope. The murmur was not to be heard with every cardiac beat; it

might be heard with a few strokes, then not for some beats, then might be once, and so on, with a variable appearance and disappearance. The whole duration of this sound was variable, differing in different cases, say from two days to three or more, mostly about a week, rarely audible up to and on day of discharge. In one case I was fortunate enough to be able to listen during a second stage labour pain, and then this murmur was noted to be constant with every cardiac stroke during that pain. If this observation be correct (and I was particularly struck with the absence of any vitiating circumstance), it seems to show that the bruit was in some manner in part at least dependent on a robust action of the right heart, the patient holding her breath during the pain. The seat of the murmur would about correspond to the "white patch" so often found on the right ventricle. I have no post-mortem evidence to offer on this point. My experience enables me to say that in all cases this murmur was a temporary phenomenon. The bruit was detected in 29 cases out of 111; it was the sole murmur in 22; it was associated six times with a murmur of the first sort; twice with one of the third sort.

I have stated my belief that the murmur is in some way related to a rubbing of the visceral over the parietal lamina of the pericardium, which laminæ may or may not be altered in some degree in structure.

Dr. John Williams and Dr. Champneys, I understand, were also of opinion that this sound owned an exocardial friction mechanism.

The arguments in favour of this view were its superficiality, its loudness, its limited area of audition and its high-pitched stiffish quality. None of these signs nor their combination is considered to be sufficient to diagnose exocardial sounds, for endocardial murmurs may undoubtedly possess these characters. A phase in the existence of this murmur in some cases was a curious "click-like" attendant on the true first sound of the

heart, this phrase used here by Dr. John Williams well expresses this occasional feature.

I am under the impression that this murmur has not been previously described in the puerperal state.

The Third Sort of Murmur.

In many respects this class is a heterogeneous one; but there is a sufficiency of common elements to warrant a grouping of the individual cases under one description. This murmur was remarkable for its loudness; its intensity was much greater than either of the others. In loudness the three murmurs may roughly be said to form a series. The first murmur may be likened to a constant galvanic current; the second to the shock of a slowly interrupted galvanic circuit; the third to the shock from a rapidly interrupted Faradic machine. They resemble these modes of electricity both in loudness and duration and in other points. This third sort of murmur was much more transitory than the second kind, and here again the serial graduation of the murmurs is remarked; so that the first sort was much more likely to be heard with every cardiac beat than the second, and the second than the third.

The duration or number of days on which the murmur was to be heard varied in like manner; speaking generally, the first sort was of longest course, the second sort next, and the third sort possessed least duration, being seldom heard for longer than three days.

These statements hold true in the main; they serve to convey as well as I am able in what way the murmurs differ from and resemble one another.

The murmur was heard 15 times out of 111; its site was very variable, but mostly basic; 2 were heard over the mitral area, 6 over the pulmonary, 6 over the aortic, 1 over both pulmonary and aortic. As a class, with only one or two exceptions, the area of audition of

this murmur was quite as limited as the murmur of the second kind. This was the murmur of all others which was mostly in relation with excited action of the heart due to pyrexia or mental emotion, or possibly other causes singly or combined. In several cases the murmur only existed when the temperature was raised, but this was not the case in all. In the most marked case of all the temperature was normal, but the patient was extremely pale, and had been so since a child. Besides pyrexia, excitement and anæmia, this murmur was very frequently associated with râles in the chest of medium size, and with a cough, but no sputa.

The quality of the sound evolved varied a good deal. In many the sound was very much like that elicited from scratching a piece of silk tightly held with the nail, or like the sound due to pressure on the carotid artery in anæmic cases, or like that heard in many cases of old phthisis over the apex of the lung where the subclavian artery is bound down by fibroid thickenings. In other cases the sibilant râle was almost exactly imitated; in one case the sound was like the sound of "crepitation," of dry and rather coarse characters.

What is the mechanism of these murmurs? All the murmurs were distinctly audible when the breathing was restrained; it seemed doubtful to me whether the varying amount of air in the chest exercised much influence on their production or intensity. Opinions were certainly divided as to their mode of generation. Perhaps this is the expression of the proper attitude to be adopted towards all questions relating to the genesis of any sound.

Dr. Champneys may have been right in some cases in supposing that the vigorously-acting heart was able with each stroke to force some air out of a pulmonary lobule or pulmonary lobules with sufficient force through a bronchiole constricted in some mode (probably from mucus, seeing the frequency of cough and râle in many cases) as to originate a sound of cardiac rhythm. Dr. John Williams may have been right in some cases in

supposing that they enjoyed a friction origin, but my inclination is to that standpoint that the murmurs were chiefly due to the blood condition plus the robust action of the heart, and I would here state my opinion that pyrexia may so alter the molecular structures in a short time as to render the production of sonorous vibrations remarkably easy. In two of the cases the rib cartilages were noted as quite yielding, and in these the murmur was, as regards its intensity, distinctly under the control of pressure through the stethoscope. One characteristic of this class must be again alluded to—important in any discussion of their mode of production—that was the extremely "capricious" mode of appearance and disappearance; this murmur was more "capricious" than either of the others.

The influence of posture on the production of all three kinds of murmur was early found out; it should be in every case the dorsal recumbent, arms to the side and head with shoulders "square." This important point was fully known to earlier writers as Dr. Matthews Duncan informed me.

A knowledge of the occurrence and features of these murmurs will prove of great value for many reasons, of which only the chief can be mentiond here. It is right that we should be acquainted with their existence from the point of view of mere knowledge, even supposing that they had no other bearing.

I was called to a woman in child-bed out Kennington way, in my capacity of House Physician to the General Lying-in Hospital. The patient was ill, with severe abdominal pain and some fulness of abdomen, with thickly coated tongue; nothing pelvic apparently; had vomited; was inclined to be delirious; temperature 102°.

On listening over the præcordia, a murmur of my first sort, blowing, soft and medium pitched, was detected loudest about fourth left space close to sternum, conducted up to pulmonary base and outwards to left apex. There was no history of rheumatic fever; the patient had had seven

children, and had always enjoyed fair health. This case happened in early part of June, 1881. It would have been easy to have thought the case one of puerperal endocarditis. There was no albuminuria (catheter). The murmur was not found a week later; the patient made a good recovery.

Dr. John Williams related to me an almost similar case, in which his opinion was sought. This case was, no doubt, a murmur of the first sort. The medical attendant, an able practitioner, was distinctly under the impression that endocarditis had been set up. The murmur cleared up with the exception of a slight pulmonary systolic when I last heard of the case.

The patient recovered, but this is not the correct expression to use; the fact is that the majority of these patients know nothing about their hearts, this organ does not, as a rule, trouble them. In reality, it is the clinical physician rather than the patient who is apt to get alarmed at the presence of these murmurs. For this reason then, if for no other, I have thought it fitting to make my observations known.

The observations recorded in the early part as well as throughout this paper are valuable as tending to explain or be explained by the normal cardiac and circulatory conditions of the puerperal state. My observations lend support to those who consider that the tension in the pulmonary artery is increased in the early part of child-bed.

How that increased tension is brought about is quite another matter. Invocation of the reflex theory of nervous action would not be a difficult matter, and this theory would lend itself with much passive plasticity to explain the facts. It would be easy to say that contraction of the uterus causes, reflexly, contraction of pulmonary arterioles. Yet it would be extremely difficult to prove anything especially in such a complicated machine as the puerperal patient, where various and varying conditions vie with each other for recognition and a place.

Summary.

My chief conclusions may be stated in a few propositions:

Systolic murmurs are of very frequent occurrence over the præcordia of the lying-in woman (75 per cent.).

These murmurs are arranged into three sorts:

No. 1 is "endocardial-like," is conducted to a variable extent, may be heard over any part of the area of the præcordia.

No. 2 is "friction-like" and non-conducted, is of very constant site (just above and to *left* of xiphoid cartilage).

No. 3 is very loud, is of very curious quality, is very capricious, and non-conducted.

The most numerous of the first sort are loudest over the right ventricle *about* the fourth left space, close to the edge of the sternum.

The mechanism of this murmur is especially doubtful. The view of Letulle that the murmur represents tricuspid regurgitation is not supported by any facts beyond and outside of the presence of the systolic murmur.

The tension in the pulmonary artery is believed to be increased in the early part of child-bed.

The "right heart" is believed to be over-acting in the early part of child-bed.

These physiological factors of the puerperal state are intimately associated with the explanation of the abnormal sounds heard over the heart in child-bed. Special stress is laid on the fact that the great bulk of the murmurs are situate over the right heart.

The murmurs are for the most part not the expression of serious mischief; they are "functional" murmurs; the medical attendant need not be alarmed at their appearance in the great majority of cases.

TWO CASES OF UNREDUCED
AND
TWO CASES OF REDUCED
DISLOCATION OF THE HIP.

BY

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THE first three of the following cases have special value of their own :—the third and fourth corroborate the opinions expressed in my paper on "Dislocations of the Thigh," in the sixtieth volume of the Medico-Chirurgical Transactions.

CASE 1. *Impacted fracture of the neck of the right femur with old unreduced dislocation of the right hip.*—James H—, æt. 75, about three hours before admission into the Middlesex Hospital on August 12th, 1881, slipped down on some stone steps, falling upon his right hip.

On admission the patient could not move the right thigh, which was somewhat flexed and slightly adducted, though the leg and foot were everted. The whole limb was shortened.

Manipulation caused so much pain that an anæsthetic was given before making further examination. The trochanter was found less prominent and higher than natural. Behind the trochanter the head of the bone could be felt on the dorsum ilii, but it gave the impression of being neither so round nor so smooth as normal. Passive movement was very limited. There was swelling of the upper part of the thigh from extravasation. The limb was steadied in a long splint and the patient put into bed. Subsequently we ascertained that many years ago he had dislocated the same limb, that the dislocation was not reduced, and the limb had ever since been deformed and shortened, but that the foot had not turned outwards *before* the accident which led to his coming into the hospital.

On August 17th I again examined him under an anæsthetic, and whilst trying, slowly and gently, to extend the limb, a crack was heard by those around, and at the same moment I felt the femur give way at its neck. The limb could now be placed parallel with the other, and on letting it go all the symptoms of fracture of the base of the cervix femoris became apparent. The Liston splint was reapplied, and moderate extension was kept up by means of a perineal band.

On October 10th he was discharged, wearing a plaster bandage, with the fracture united and the limb in a good position.

In this case I believe that there had been an impacted fracture at the base of the neck of the femur, and that by manipulation it became unimpacted. Thus is explained the association of flexion and adduction of the thigh with eversion of the limb and the presence of the head of the bone on the dorsum ilii. Advantage was taken of the fracture to correct the deformity of the dislocation, and the patient was thereby placed in a better condition than before his recent accident.

Though I would not fracture the cervix femoris intentionally in a case of old dislocation, I would suggest in such cases, if the deformity is marked, the shortening

considerable, and the mobility slight, and if the patient is not very old or otherwise a bad subject for operation, that subcutaneous division of the neck of the femur might safely and advantageously be performed with a view to correct the more serious inconveniences of dislocation.[1]

The next case shows how much may be done in a young and energetic person by perseverance and exercise, towards minimising the drawbacks of an unreduced luxation of the hip.

Case 2. *Unreduced dorsal dislocation of the hip with remarkably good movement of the limb.*—Francis F—, æt. 25, a discharged soldier, was admitted into the Middlesex Hospital in December, 1881, having been sent to me by my friend and colleague, Dr. Fowler. The patient, a well-educated and very intelligent man for his station in life, wrote the following account of himself while in the hospital:—"In the early morning of October 12th, 1880, at the Manoorie Barracks, Poona, I fell from a height of twenty-five feet by walking, when in a state of somnambulism, through a window which opened down to the floor. I was found lying on the ground where I had fallen, and was carried to hospital about 2.30 a.m. in an unconscious state, and so remained until the next morning. The surgeon-major, whose patient I became at first, formed the opinion that there was fracture, with displacement of the trochanter upwards and outwards. For ten weeks hot fomentations were applied to my hip, and during this time the limb became short; then a long splint was applied for four days and four nights, and thus the knee, which was becoming bent, was straightened. I was examined by four or five surgeons at different times; one

[1] I was not aware when this paper was read that Dr. H. G. Rawdon had *excised the head of the femur* in a case of unreduced dorsal dislocation of the hip, the result of typhoid fever (*vide* 'Liverpool Medico-Chirurgical Journal,' January, 1882, p. 22). I have since witnessed the performance of the same operation by Mr. W. Adams for unreduced dorsal dislocation coming on during fever. In each case the operation was followed by an excellent result.

diagnosed dislocation of the femur with the edge of the socket chipped. After this an operation was performed by pulleys used across the other thigh, but without any result. They gave me chloroform for this, but did not get me completely under it. Other attempts were made, both with and without pulleys, to reduce the limb. When first discharged from hospital, viz. in the early part of March, 1881, I walked very lame, but after taking to swimming I gained more and more use of the limb. On June 12th I arrived at Netley Hospital with other invalids, and was examined on several occasions by Surgeon-Major Tobin, A.M.D., and Surgeon-General Longmore, C.B., whose opinion was dislocation of the femur and chipped socket."

This account is confirmed by the reports which I have received from the Secretary's office at Chelsea Hospital, and from Professor Longmore at Netley.

The notes taken of his condition whilst in the Middlesex Hospital are these:—There is 1⅝ inch shortening of the limb, and the top of the trochanter is this much above Nelaton's line. On forced passive flexion and circumduction there is fibrous creaking felt and even heard. The head of the femur is felt on the dorsum ilii, and between the head and the trochanter the neck is easily traced. There is felt the characteristic hollow behind the large vessels in the groin from the absence of the resistance offered by the head of the femur when in its normal place; the edge of the acetabulum itself can be felt at the top of the thigh in front if the fingers are well pressed in. The buttock is flattened, and the fold of the nates a little fallen.

The voluntary movements of the limb are almost perfect. When the patient stands with the sole of the foot flat, but a little raised, he looks quite straight, the shoulders and hips being level. Whilst standing he can flex his thigh beyond the right angle, and whilst lying down and stripped he can with the aid of his hand make his knee touch his shoulder of the same side. Standing

he can separate his legs a distance of upwards of forty-eight inches; lying down the sound limb can be abducted thirty inches before the pelvis moves, and the dislocated one twenty-three inches.[1] Adduction and rotation are almost if not quite perfect; the dislocated limb can be swung across either in front of or behind the sound one. Extension is complete, and when he walks the toes are directed nearly if not quite straight forward. He walks, however, with a peculiar gait, which is something between a roll, a twist, and a jerk. The only other defect is an inability to commence to stand on the displaced limb alone, but having once balanced himself he can continue for a little while to stand upon it without support, but not very steadily.

I made an unsuccessful attempt under chloroform to reduce by manipulation. I tried the usual movements twice, but the bone did not seem to approach the socket, and a good deal of fibrous cracking was produced. The interval of fourteen months since the accident, and the excellent movement of the limb, prevented me from making any very forcible efforts at reduction.

At my first examination of the patient I expressed the opinion that there had been fracture of the rim of the acetabulum, and subsequently direct dislocation on to the dorsum ilii. My reasons for so thinking I stated to be: (1) the nature of the accident, viz. falling from a height upon the feet whilst walking with the limbs, stiff as they usually are, in a state of somnambulism; (2) the absence immediately after the injury of the characteristic symptoms of dorsal dislocation, and their slow development whilst the patient was lying in bed; (3) the suspicion which the first surgeon who examined him expressed, that there was a fracture with displacement upwards and outwards of the trochanter major; (4) the free mobility of the thigh, which is more likely to be acquired if the head of the

[1] The amount of abduction was measured by the distance between a straight line through the middle of the body continued to the feet, and the internal malleolus of the abducted limb.

femur rests immediately on the dorsum ilii than if it mounts upwards over the obturator internus and pyriformis muscles.

The case offers I think an illustration of the way in which dislocation of the hip may be overlooked in spite of careful examination soon after the accident. If violence which would cause dislocation when the thigh is *ab*ducted, be inflicted on a limb which is not *ab*ducted but adducted and extended or even partly flexed, the margin of the acetabulum must break before the head of the femur can be displaced from its socket. And unless the violence which breaks the margin of the socket is also sufficient to force the head of the femur at the same time quite out of the acetabulum, the head may be retained against the broken edge but still within the capsule, being caught as it were between the detached fragment and capsule on one aspect and the broken socket on the other. In this position the head of the femur is ready at any moment to be jerked, or gradually and after a longer or shorter time to be drawn by muscular action out of the socket on to the dorsum ilii or on the ischium as the case may be. The practical lesson such a case seems to teach is to apply a splint and keep up extension in any person in whom the nature of the accident or the condition of the limb suggests, even remotely, the possibility of fractured rim of acetabulum.

The extraordinary motion of which the limb of this man is capable is no doubt in part due to his youth and athletic activity (he was instructor of gymnastics), and to his persevering efforts to regain the use of it. But it is in part, I think, the result of the character of the luxation. The head of the femur, being in immediate contact with the ilium, has doubtless by this time formed for itself a shallow cavity on the bone under cover of the muscles; and the muscles by strapping the head down assist in steadying it; whilst the neck of the femur is controlled by the detached edge of the capsule (*i.e.* by the margins of the aperture through which the head of the bone escaped).

Whereas when the head of the femur rises over the rotator muscles, as it does in indirect dorsal dislocations, *i.e.* dorsal dislocations unaccompanied by fracture, then the neck of the femur is wound upon and bridled by the untorn segments of the capsule.

CASE 3. *Thyroid dislocation of the femur; reduction by manipulation.*—Arthur S—, æt. 29, a fireman, was driving a fire-engine, and on entering a street which was partly dug up, some one flashed a light across the road and startled the horses. The engine came violently in contact with a lamp-post, and the patient was thereupon thrown from his seat on to the pavement, forwards. He alighted on his knees, "chiefly on the right, and on the helmet and stretched out hands." He was taken at once to the Middlesex Hospital and admitted on October 5th, 1879.

The right thigh was flexed on the trunk to about 60° and could not be straightened; the foot was slightly everted; the trochanter major was sunken, and nearer the central line of the body than the trochanter of the other side. The head of the femur formed a hard projection at the junction of the perineum and thigh, and could be felt resting against the arch of the pubis at the junction of the rami of the pubes and ischium. It could be made to rotate in its new situation by rotating the limb. The tendon of the adductor longus was stretched very tightly across the front of the head of the bone. On placing the other limb in a position as nearly as possible similar and then measuring both limbs from the anterior spine of the ilium to the top of the patella, about 1¼ inches of shortening was found on the dislocated side. Passive flexion was free, and slight voluntary flexion possible; but extension was impossible and any attempt at it caused severe pain. There was a large patch of ecchymosis on the inner side of the right knee, and a smaller and less well marked one on the inner side of the left. The patient was brought fully under ether and reduction by manipulation attempted by

the house surgeon. The leg was flexed on the thigh, and the thigh on the trunk; the knee pushed as far as possible over to the middle line, and the limb circumducted inwards and quickly extended. When let go the thigh was found to be dislocated on to the dorsum ilii.

The manipulation proper for dorsal dislocation was then employed, but during circumduction the house surgeon kept up extension on the thigh with his hand under the ham. With a distinct snap the head of the bone re-entered the socket and the passive movements of the limb were at once natural. At the end of the third week the patient was discharged quite well.

In this case the dislocation was of the thyroid variety, but the head of the bone was displaced further inwards towards the perineum than is usual, though it did not absolutely reach the perineum. It is commonly stated that in thyroid luxations there is from one inch and a half to two inches of lengthening of the dislocated limb, but the general accuracy of this statement may be well questioned. The lengthening, I believe, is more apparent than real, and is due to the very considerable tilting of the pelvis, which in this variety of luxation is very marked and altogether more extreme than in any other form of hip dislocation. Probably the *real shortening* in this case was greater than usual because the head of the bone had passed to the extreme verge of the thyroid foramen and was close to the perineum. Mr. Holmes[1] has described a case of obturator dislocation in which the injured limb, though it looked longer than the other was really shorter; and Mr. Rivington[2] another, in which, though there was apparent lengthening, there was real shortening of from one half to three quarters of an inch.

CASE 4. *Dorsal dislocation of the hip; reduction by manipulation.*—John P—, an active labourer, æt. 24, was admitted into the accident ward on January 5th, 1882,

[1] Holmes, 'Med. Times and Gazette,' Oct. 27th, 1877, p. 455.

[2] Rivington, 'Lancet,' vol. i, 1878.

with well-marked dorsal dislocation of the right hip. He was engaged in picking away earth for the foundations of a wall when a quantity of the earth fell upon him, and partly buried him in a sitting or, more properly speaking, a squatting position. When extricated, his right lower limb was painful and useless.

On admission, immediately after the accident, he was in considerable pain; the right limb was 1⅛ inches shorter than the left, and presented all the symptoms of a dorsal dislocation, except that, as the leg was somewhat flexed upon the thigh, the inverted right foot was at some distance from the left foot. This position of the leg was doubtless the result of the tension on the hamstring muscles and on the sciatic nerve. The trochanter on the right side was nearer by half an inch to the anterior superior iliac spine than on the left. The hollow behind the vessels in the groin, as felt by dipping the fingers, was extremely well marked. The head of the bone was very plainly felt in its new position on the dorsum.

A mixture of chloroform and ether was given at once, and the house surgeon undertook at my request the reduction by manipulation. The first attempt was unsuccessful owing to the outward circumduction and rotation not being quite decisive enough. A second attempt, during which I kept my fingers on the head of the femur, and when it had reached the lower edge of the acetabulum, gave it an inward and upward pressure, was quite successful; the head of the bone re-entering the acetabulum with a distinct snap. He made a quick recovery.

In this case the ease with which the head of the bone could be felt travelling downwards at the back of the acetabulum, and that too without any marked resistance being experienced by the operator, amounted almost to an actual demonstration that the bone was retracing the course along which it had been displaced.

The facility with which the head of the bone reaches the dorsum from a position below the acetabulum is

shown by Case 3. The first attempt at reducing it by manipulation converted the thyroid into a dorsal luxation. Indeed, the cases in which either pubic or thyroid dislocations are converted into posterior dislocations afford another argument in support of the theory that in all dorsal and so-called ischiatic luxations (uncomplicated by fracture) the head of the bone reaches its final position indirectly and secondarily, and after leaving the acetabulum in a downward direction. In escaping from the socket the head of the femur passes through the thin part of the capsule, and may take an inclination backwards towards the tuber ischii, or inwards towards the pubes, according to circumstances. But its course is essentially a downward one in the first place; whilst the rent in the capsular ligament, though usually limited to the thin portion, varies, as I have elsewhere plainly stated, with the degree of upward or backward displacement.

And here I would reply to one or two of the remarks on this subject in a paper in the sixty-third volume of the Society's Transactions.[1] It is unnecessary for me to allude to several of the criticisms contained therein; but with reference to the statement that, "in order that the head of the bone may be thrust through the inferior portion of the capsule, over the cotyloid notch, the thigh must be *abducted* in the extended position," I would reply that extension forms no part of the necessity. And when the writer adds that "this is totally at variance with the opinions of Sir Astley Cooper, Hamilton, and Bigelow, who teach that flexion with adduction is the position in which dislocations backward occur," my answer is that, setting aside the question whether this is or is not a correct summary of the opinions of these distinguished men, the object of my paper was to show that this opinion, so far

[1] Two cases of direct dislocation backwards of the femur, with fracture of the rim of the acetabulum; with remarks on the mode of production of dislocations backwards; by Fred. S. Eve, F.R.C.S., 'Med.-Chir. Trans.,' vol. lxiii, p. 51, 1880.

as regards *adduction*, is generally, if not always, wrong. Also I cannot agree with the writer's views of abduction, nor with the conclusions he draws therefrom; and I believe that a very simple experiment would convince him that he was in error when he argued "it will be found impossible to straddle the legs widely apart, that is, abduct them, while the feet are inverted."

Of the nine museum specimens tabulated in the paper referred to, six of them had been described by me; one in my published paper and five in some MS. notes of all the specimens of hip dislocations contained in the London museums up to January, 1877, and which I lent to the writer before he had prepared his paper for the Transactions. All these specimens had been well considered before I ventured to publish my opinions. The other three cases in the table were added to the St. Thomas's and St. Bartholomew's Museums after the date of my paper. Two of them support my views, as they are direct dislocations with fracture of the acetabulum. The third is Sir W. MacCormac's. I cannot admit, for reasons I have elsewhere stated, that either of the cases which the author calls *direct dislocations without fracture* in the least invalidates my arguments—arguments which were based, not on experiments alone, but on anatomical grounds, on the consideration of the accidents which cause dislocation of the hip, and on the mechanism and success of the method of reduction by manipulation.

It is inconsistent to speak of a dislocation on to the tuber ischii, or one in which the head of the bone leaves the acetabulum below the level of the obturator internus tendon, as it did in Mr. Wormald's and Mr. Adams's[1] cases, as examples of "direct backward dislocations."

[1] Mr. Adams's specimen has recently been dried and added to the Hunterian Museum. It was for a very long time preserved wet in the preparation room, but never placed in the museum. I saw it in its wet state. The capsular ligament was torn in exactly the manner I have described as being commonly the case. No specimen could possibly afford stronger confirmation of my views.

Direct they can be, but they are necessarily "downward and backward," not simply backward in their directness and direction; and to reach its new situation the head of the femur passes over the shallow part of the edge of the acetabulum and through a rent in the thin part of the capsule.

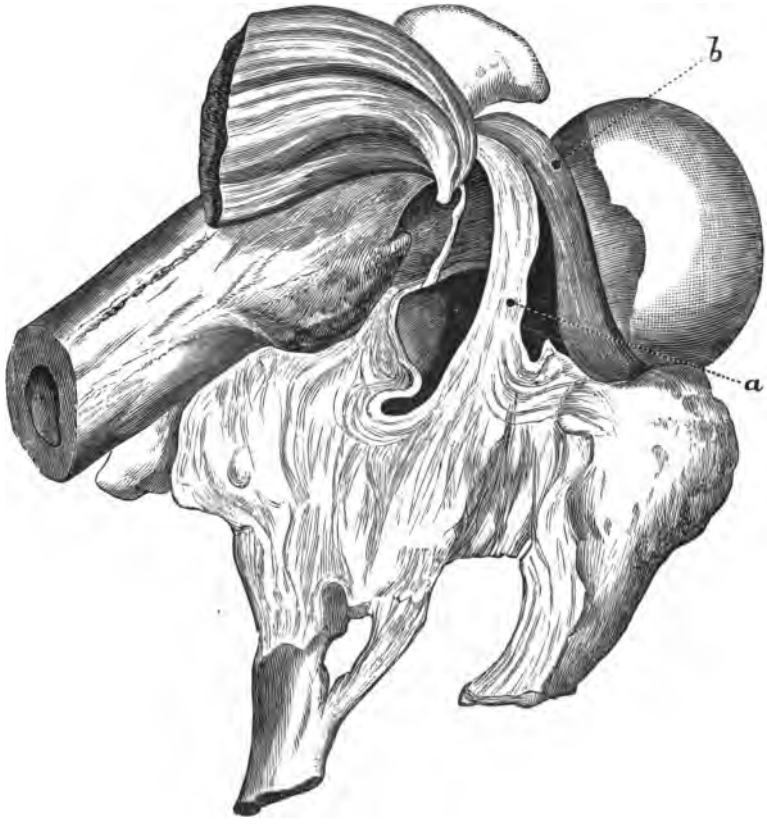
This is what I have contended for:—(1) that in all the ordinary dislocations backwards *without fracture*, whether they are spoken of as "dorsal" or "sciatic," or, as Bigelow styles them, "dorsal above" and "dorsal below the obturator internus tendon," the head of the femur leaves the acetabulum in a downward direction through a rupture of the thin part of the capsule, and emerges therefore below the internal obturator tendon; (2) that herein we have the *rationale* of the success of the methods of reduction by manipulation; and (3) that *posterior as well as anterior* dislocations occur whilst the limb is abducted.

Since my former communication on this subject to the Society I have carefully searched through the Musée Dupuytren in Paris, which is comparatively rich in hip dislocations, and the museums at Montpellier, Bordeaux, and Toulouse. I had previously examined all the museums in London, and have since re-visited several of them. And this I can say is the result of my search: that, with one exception, there was no preparation in any of these museums, nor in the museums of Dublin [1] or Manchester,[2] which tends to refute in the least the above assertion, though there are several which strongly confirm it. The exception is Sir W. MacCormac's case, in which the head of the bone was forced through the capsule twice, first downwards through the thin portion

[1] To Mr. W. Thomson I am indebted for information respecting the specimens in the following museums in Dublin, viz. Trinity College, College of Surgeons, the Richmond Hospital, Mater Misericordiæ and Dr. Steevens's Hospitals.

[2] To Mr. Fred. A. Southam, of Manchester, my thanks are also due, for looking through the museum at Owens College.

of the ligament, which is rent in the usual manner, and then through the back of the capsule above the tendon of the obturator internus. It is the exception which proves the rule. The specimen is a very remarkable one and shows the two openings, separated by a broad unbroken band of the ischio-femoral portion of the



a. Unbroken band of the capsule.
b. Obturator internus muscle.

capsule, as is seen in the very accurate drawing by Mr. Noble Smith, and I am indebted to the kindness of

Sir W. MacCormac, and of Mr. Stewart the curator of St. Thomas's, for permission to draw it.

The man was seventy years of age and the dislocation occurred thus :[1]—"Whilst at work in an iron foundry, a metal casting, ten hundred weight, fell upon him. The mass was at the time partially suspended in a crane, one end resting upon the ground, when the tackle gave way and the man was thrown violently on to his knees upon a wooden block, between which and the metal the abdomen was forcibly compressed. The patient, an intelligent man, described how the casting first struck his hip, and that he subsequently felt himself twisted round towards the prone position during the act of being crushed." The effect of the blow was to dislocate the left hip, and the effect of the compression was to cause several feet of intestines to exude through a rent in the walls of an old inguino-scrotal hernia.

If the specimen and the description of the accident are taken together, it is clear that the head of the bone was first of all driven through the lower opening in the capsule by the blow on the hip which knocked the man on to his knees; but afterwards, whilst the lower end of the femur was resisted by the ground, and as the metal casting twisted his body round towards the prone position and crushed it downwards, the head of the femur was, so to speak, overtaken by the pelvis and forced straight backwards through the upper opening in the capsule between the pyriformis and internal obturator muscles.

[1] 'St. Thomas's Hospital Reports,' vol. ii, p. 143.

ON THE VARIATIONS

IN THE AMOUNT OF

SULPHOCYANIDE OF POTASSIUM

IN THE

SALIVA OF PERSONS AFFECTED WITH DIFFERENT DISEASES.

BY

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It being known that both the colouring matter and the salts of the bile can be occasionally detected in the saliva in cases of disease, the thought was naturally suggested that perhaps, under normal conditions, the saliva might contain some substance resulting from the decomposition which the biliary acids are believed to undergo. It is well known that the saliva contains sulphocyanide of potassium, which is easily reognised by the red colour produced on the addition of a persalt of iron; and as the sulphocyanide is met with only in the saliva, as it contains sulphur in an unoxidised form, and as it is known to vary greatly in amount in different cases, it was selected for observation.

The amount of the sulphocyanide is so small that a quantitative test is a matter of great difficulty; it was

therefore thought necessary to trust to the depth of colour produced by the addition of a weak solution of perchloride of iron. The saliva of each person examined was collected as it flowed from the mouth for about two minutes; and seven minims of a solution of half a drachm of the iron to an ounce of water were added, and the colour thus obtained was compared with a scale of colours prepared in the following manner:

A quantity of saliva was collected from a number of healthy persons, and the tint produced by the addition of the perchloride of iron was taken as the normal amount; a portion of the same saliva was then evaporated to a quarter and another to half its bulk, and the colours produced in these respectively were assumed to represent four times and twice the normal quantity of sulphocyanide; while two other portions diluted with water to twice and four times their amount were taken as representing half and a quarter of the normal quantity of the salt.

In this way I examined, during the course of many years, the saliva of nnmerous persons who consulted me for different maladies, recording also the symptoms of which they complained, and in most cases the results of the treatment adopted.

These observations were so numerous (probably many thousands) that I selected for analysis only those in which the amount of the sulphocyanide was much below or much above the normal standard. In the following pages these are described as "private cases."

In order to check these observations I requested Dr. Needham, at that time house physician to the London Hospital, to examine the saliva of a number of patients who were under treatment in the wards, and two years afterwards a similar inquiry was carried on by Dr. Bedford Fenwick in the same hospital.

The cases thus obtained, numbering between 200 and 300, have been analysed together, and are described as "hospital cases."

As it has been asserted by some physiologists that the

presence of the sulphocyanide of potassium in the saliva is accidental, and is the result of decomposition set up by decayed teeth, it seemed desirable, in the first place, to ascertain how far such an opinion is supported by facts.

Careful inquiries were made as to the state of the teeth in 87 cases in the hospital, and with the following results. The teeth were quite perfect in 18 per cent. of those whose saliva contained a normal or excessive amount of sulphocyanide, and in only 14 per cent. of those in whom it was deficient. One or two teeth were carious in 47 per cent. of the former and in 44 per cent. of the latter; whilst many were decayed in 42 per cent. of those in whose saliva the sulphocyanide was below, and in 35 per cent. of those in whom it was normal or excessive. It is evident, therefore, as the salt presents itself in excessive quantities where there is no decay of the teeth, and is as often deficient as superabundant where many teeth are carious, the opinion before mentioned must be incorrect.

Others have attributed the presence of sulphocyanide to the smoking of tobacco, and the habits of 213 patients in the hospital were investigated as to this point. As the greater number had been confined to their beds in wards in which smoking was not allowed, only a few of them had latterly enjoyed the opportunity of using tobacco. On analysing these cases it appeared that of those whose saliva contained an excess of sulphocyanide 6 per cent. only had latterly smoked; of those presenting a normal quantity, only 2 per cent.; whilst of those in whom there was a deficiency, 8 per cent. had latterly consumed tobacco, and one represented himself as an excessive smoker. It is evident, therefore, that no material influence is exerted by the use of tobacco, although my own impression, before analysing these cases, was that the sulphocyanide is usually present in greater quantity in the saliva of those who smoke than of those who abstain from tobacco.

I propose, in the first place, to examine only those

cases in which the sulphocyanide was deficient, and to see what conclusions can be drawn from them.

One of the most striking points that presents itself in these cases is the frequency with which it is below the normal amount in jaundice arising from obstruction. Thus, of 23 persons admitted into the hospital with this disorder, it was deficient in 18, and in many of them not even a trace of colour could be obtained by the addition of the perchloride of iron. It 2 the saliva was normal, but one of these had so far recovered as to have been placed on full diet, and in the other post-mortem examination proved the jaundice to have resulted from the rupture of a hydatid cyst into the ducts of the liver, the opening of the common duct being only partially obstructed, and bile being present in the duodenum. In 1 case out of 3 in which the sulphocyanide was in excess the jaundice was probably due to partial obstruction of the ducts of the liver by carcinomatous growths; the histories of the others could not be obtained.

From these facts it would seem probable that in order that the sulphocyanide should appear in the saliva, the bile must be able to enter the duodenum, and this seems to be supported by 2 cases admitted into the London Hospital, in which large quantities of bile were discharged through the lungs, probably from rupture of hydatid cysts of the liver; and in both of which scarcely any colour was developed in the saliva by the addition of perchloride of iron.

Pointing to the same conclusion is the circumstance that in some cases of jaundice the depth of the colour of the stools varies with the amount of the sulphocyanide. Thus, a lady had suffered three weeks from jaundice, arising apparently from hypertrophic cirrhosis; the skin and urine were deeply tinged, the stools white, and the saliva free from sulphocyanide. She was treated with perchloride of mercury, and in three weeks the stools became partially coloured and a slight tinge was produced in her saliva by the addition of perchloride of iron.

In two weeks afterwards diarrhœa occurred, the stools being deeply stained with bile, and although the skin and urine remained yellow, the saliva contained an abundance of the sulphocyanide.

Case 2.—A gentleman had been for three months deeply jaundiced, the saliva being −2. In two weeks afterwards the jaundice had greatly lessened, the stools had become brown coloured, and the saliva was +2. Two or three weeks after this he had a severe attack of pain in the abdomen, with an increase of the jaundice, and the sulphocyanide again became deficient. When I last saw him he had been much better, and the sulphocyanide was again +2.

In severe cases of jaundice arising from obstruction, and it must be remembered that only severe cases are admitted into the hospital, the deficiency of the sulphocyanide has been usually greatest at the beginning of the disease. Thus, in all excepting one, which was of three months' standing, where there was a complete absence of the sulphocyanide (− 8), the saliva was examined within one week of the coloration of the skin, whilst in those whose average duration had been five weeks the saliva was −4.

This rule, however, does not hold good for those temporary cases of jaundice following severe abdominal pain, and usually supposed to arise from gall-stones. In two such instances the saliva was normal, and in each the jaundice passed away in a day or two. If these observations should be confirmed by subsequent inquiries we shall have in the saliva a valuable means of prognosis; for when the sulphocyanide is completely absent at the outset of the disease, the case will be probably more obstinate than where some can be discovered. In chronic cases (for example, after three months' duration) I have generally found that when the sulphocyanide was *permanently* absent the prognosis was most unfavorable, but when its presence could be still discovered the obstruction was incomplete and recovery often took place.

Where there is no obstruction to the entrance of the bile into the duodenum, one of the chief circumstances which seems to determine the amount of the sulphocyanide is the quantity of food, and especially of animal food, that is taken into the stomach and digested. For example, in all the cases of obstruction of the œsophagus I have examined, there has been from the first a marked lessening in the amount of colour produced by the addition of the perchloride of iron, and this has progressively increased as the difficulty of taking food has augmented.

In eight cases of cancer of the stomach the sulphocyanide has been very scanty or entirely absent.

In connection with this it is worthy of remark that physiologists have come to the conclusion, from experiments on animals, that the amount of the bile secreted is mainly regulated by the quantity of food that is taken, and that when food is altogether withheld the secretion of bile rapidly decreases.

Of course persistent vomiting must lessen the amount of food digested as certainly as if only a small quantity were given, and we find that in 111 of the "private cases," where there was a marked deficiency of the sulphocyanide, 29 were suffering from a more or less constant rejection of the contents of the stomach.

Long-standing diarrhœa and dysentery seem also to act like vomiting, in lessening the amount of the sulphocyanide; for in 23 out of 111 "private cases," where it was deficient, one of these conditions was present, but a temporary attack of diarrhœa seldom produced much alteration. It is interesting to observe that Dr. Rutherford, when reviewing the results of his experiments on the secretion of bile in dogs, asserts that "we have, however, found several drugs that have an indirectly depressant action; thus, when the intestinal glands are excited to secrete, there is an indirectly depressant effect on the liver, whereby the bile secretion is lessened. We invariably observed that while slight purgation by a purely intestinal irritant scarcely, if at all, depressed the secretion of

the bile, powerful purgation produced a very marked effect."[1]

As the sulphocyanide has been shown to be deficient whenever the supply of food has been small, it might be surmised that this condition would also present itself in atonic dyspepsia, in which disease the digestion of the food is so imperfect. This is in fact by far the most frequent cause, for 39 out of 111 "private cases" were instances of this disorder. They were all very severe cases, the appetite being bad in almost all, and in many the sensation of hunger being entirely absent, or replaced by loathing of all food.

It is probable that feebleness of the digestive powers is also the most common cause of the deficiency of the sulphocyanide in various chronic maladies; for in 84 persons presenting this condition, in whom this point was carefully investigated, the appetite was stated to be bad in 55 cases, moderate in 12, and good in only 17 of the whole number.

The general nutrition of the body was also very defective; for of 53 cases, where the sulphocyanide was below the normal amount, 40 are stated to be thin, 3 moderately stout, and only 10 stout. In some of the latter the deficiency in the sulphocyanide was only temporary, and of the remainder, 4 are stated to be losing flesh.

There is, however, one class of cases which cannot be referred either to a want of food or to a deficiency of the digestive power. In every severe case of lead poisoning the sulphocyanide was absent or present in very small quantity, the deficiency persisted so long as the symptoms were urgent, and it was succeeded by an excess as soon as convalescence began.

This was so invariably the case that I have been in the habit of employing it as a test of the condition of the patient, and have found it valuable in diagnosis and prognosis.

[1] Prof. Rutherford, 'On the Action of Drugs on the Secretion of Bile,' p. 170.

The cause of this circumstance seems capable of explanation in two different ways. At first I supposed that, as the saliva was always very thick and tenacious, the lead acted as an astringent to the salivary glands, but I found that the deficiency of the sulphocyanide remained, even when these were stimulated to increased secretion by the application of tincture of pyrethrum to the tongue. A more probable hypothesis seems to be that the lead acts as a sedative upon the liver and prevents or lessens its secretion.

This view is confirmed by the experiments of Dr. Rutherford, who states that, "It cannot fail to strike the reader as a remarkable fact that while in the long list of drugs whose hepatic effect we have investigated we have found so many that stimulate the liver, there is only one—'acetate of lead'—which appears to have a directly depressant effect."

It might naturally be expected that the secretion of sulphocyanide would be often modified by affections of the salivary glands themselves, but these glands are so little liable to disease that this seems seldom to be the case. I have, however, seen some cases of acute stomatitis in which no colour could be produced in the saliva by the perchloride of iron, but it is more probable that this arose rather from the inability of the patients to take food than from the effect of the inflammation of the mouth upon the glandular secretion.

In reviewing the circumstances that have appeared chiefly to coincide with a deficiency of the sulphocyanide in the saliva, we have found—

1. Any obstruction to the free entrance of the bile into the duodenum.

2. Any circumstance that diminishes the amount of food that is digested or absorbed, such as (*a*) œsophageal stricture, (*b*) gastric cancer, (*c*) atonic dyspepsia, (*d*) persistent vomiting, (*e*) long-standing diarrhœa.

3. The effects of lead poisoning.

The two latter circumstances are, as has been shown,

recognised by physiologists as the chief agents that in health tend to diminish the secretion of bile.

I propose now to inquire into the circumstances under which the sulphocyanide presents itself in the saliva above the normal quantity.

If the previous conclusions be correct, we should expect that an excess of the sulphocyanide would be found in conditions the reverse of those in which it was deficient: and such appears to be the case. Thus, in contrast to the figures before quoted as to the loss of appetite when the amount was below the normal, we find in 67 per cent. of the "private cases" whose saliva presented an excess that the appetite was good, in only 33 per cent. was it at all defective, and in no case was an absence of appetite a subject of complaint. Again, in opposition to the fact that most of those who had a deficiency of the sulphocyanide were thin or losing flesh, only 6 per cent. of those presenting an excess were thin, 20 per cent. were moderately stout, and 74 per cent. are recorded as "fat."

It was shown before that there is no good reason to believe that the amount of the sulphocyanide is in proportion to the quantity of bile secreted, and that this again depended in a large measure upon the amount of animal food digested and absorbed; so we may now conclude that an excess of the sulphocyanide represents an excess of nutritive material beyond what is necessary for the requirements of the system. The practical value of such an inference will be readily appreciated, and I have been in the habit of regulating the diet of dyspeptics in accordance with it. Where the sulphocyanide has been below the normal a liberal amount of animal food with alcoholic stimulants has been prescribed, but when it has been in excess a reduction in the quantity of food and stimulants, or an entire absence of alcohol, has been found to be beneficial.

We are not, however, by reference to the amount of food digested, able to explain all the cases in which the sulphocyanide is in excess, for it has presented itself in

various diseases in which the patients were restricted to a very meagre diet.

This was especially observable in acute rheumatism. Of 36 cases of this disease admitted into the hospital all but one, whose saliva was *normal*, showed a marked excess of sulphocyanide, the amount varying from eight times to twice the ordinary quantity. The depth of colour also varied at different periods of the malady; those examined within seven days of the commencement of the fever presenting an average of + 4·8, in the second week it reached + 6, in the third week it fell to + 4, and in the fourth to + 3½.

The excess corresponded, in some degree, with the temperature and the pulse; those having a saliva + 2 presented an average pulse of 81 and an average temperature of 100·6°; those + 4 a pulse of 92 and a temperature of 100·9°; whilst such as had + 8 are recorded as having an average temperature of 101·3° and a pulse of 101.

This persistence of an excess of the sulphocyanide and its gradual decrease in acute rheumatism is interesting in regard to the effects of the salicylic acid upon the disease. Although the fever and the pains of the joints rapidly subside under its use, relapses are so apt to occur that the average duration of the patients in the hospital is not materially lessened.

It would seem probable from the persistence of an excess of the sulphocyanide that the fever runs a definite course, as is the case with the infectious febrile diseases. The saliva is always very acid in acute rheumatism, and I have found the patient is liable to relapses so long as his saliva is in an abnormal condition.

It is probable that persons who have habitually a large amount of sulphocyanide in their saliva are more liable to local rheumatism than others; for amongst the "private cases" 40 per cent. of those who had an excess of it had suffered from pains of the limbs or joints; whilst neuralgic affections were more common amongst those whose saliva presented a deficiency.

All the cases of gout in the "hospital cases" had an excess of sulphocyanide, although not to so great an extent as in rheumatic fever; in the "private cases" there were 17 in whom it was above the normal, and there were none amongst those who exhibited a deficiency. The amount seems often to increase just before an attack, and I have on several occasions been able to predict a fit of gout by observing this.

In that large class of cases so commonly regarded as "gouty," but where there had been no articular affection, the saliva generally presented an excess of the sulphocyanide, and thus furnished a valuable indication for treatment. In almost every case of eczema and urticaria the same condition of the saliva was discovered, and it was also usually present in the various affections to which gouty persons are so liable.

Headache was one of the most common complaints to which those having an excess were subject, and the so-called "bilious headaches" constituted 25 per cent. of the whole number of "private cases," where this condition of the saliva was present. Persons suffering in this way usually belonged to families in which other members had suffered from gout or rheumatism; the attacks were periodical, and were often followed by a temporary improvement in health.

One tenth of those producing a deficiency also complained of "bilious headaches," but there was usually no gouty history, no relief was afforded by the attack, and the pain seemed to be of a more purely neuralgic character. The importance of such a distinction in a practical point of view need not be pointed out.

But if the sulphocyanide is so constantly in excess in acute rheumatism and gout, are we justified in regarding these diseases as produced by an excess of the sulphocyanide or the presence of some other sulphur compound in the system?

It is true that where it is in excess there is also very often an excess of lithic acid in the urine, but the excess

of sulphocyanide occurs in so many different disorders of a total different character that such a hypothesis cannot be supported.

In the early stage of almost all febrile and inflammatory affections the sulphocyanide is usually in excess. For example, in gastric catarrh this condition is almost always present; thus, 27 per cent. of the private cases who had an excess were suffering from this complaint. It might, indeed, be said that a large proportion of persons affected with chronic gastric catarrh are gouty; but the same state of the saliva was present in those suffering from recent ulceration of the stomach, although they were restricted to a very meagre diet. We have before seen that the most common complaint amongst those whose saliva showed a deficiency was an *atonic* state of the digestive organs, and it now appears that inflammatory affections of the stomach are as constantly present in those in whom it is in excess; we have therefore a means of diagnosis between these states of the digestive canal which it is so often difficult to distinguish, but in which the successful treatment entirely depends upon the accuracy of the diagnosis.

As far as my observations have gone I should be inclined to state that all previously healthy individuals, when affected with an inflammatory disorder, present an excess of sulphocyanide during the acute attack, but that this is afterwards followed by a diminution. For example, of 9 cases of acute pleurisy 4 were examined within one week of the commencement of the illness, and 3 presented it in excess, and 1 in normal quantity; 3 were examined between the second and third week, and 2 had an excess and 1 less than normal; after the third week 2 were examined, and both were below normal.

Twenty-six cases of phthisis are recorded, of whom 25 per cent. had a deficiency of sulphocyanide when examined within six months of the beginning of the illness; 58 per cent. of those between the sixth and twelfth months; and

no less than 87 per cent. of those above twelve months presented the same condition.

It was before shown that in rheumatic fever the amount of sulphocyanide bore some proportion to the height of the temperature and the rapidity of the pulse; but in phthisis the opposite is observed. Thus, of six cases below the normal amount the average temperature was 101°, whilst of seven whose saliva was normal and of six cases of excess, the average of each was 99·4. It is evident, therefore, that the formation of sulphocyanide is not necessarily the result of a high temperature; and if we bear in mind the loss of appetite and the tendency to vomiting and diarrhœa that so constantly accompany the advanced stages of phthisis, we can have little difficulty in understanding why the sulphocyanide should be deficient at this period of the disease.

In diseases of the kidneys the excess is greatest at the outset of the illness; the average duration of those presenting +8 was 5½ weeks, those +4 was 5 months, and those +2 was 14 months.

Ten cases are registered as suffering from heart disease; those under twelve months' duration had all an excess or a normal amount of the sulphocyanide; but after twelve months, where the digestive powers had become enfeebled, as is almost always the case in this malady, every case presented the sulphocyanide below the normal quantity.

The question arises whether there is any hypothesis by which we can explain the above observations. We have seen that in stout persons and in those gaining flesh the saliva usually contains an excess of sulphocyanide, and the most probable explanation seems to be that one of the functions of the liver in health is to break up any nutritive material that may be absorbed and is in excess of the requirements of the system. We know from the researches of Andral and others, that in the diseases in which an abnormal quantity of sulphocyanide has been discovered, viz. in rheumatic fever, erysipelas, and all acute inflammations, the albumen of the blood is so altered

that the quantity of the fibrin-forming material is greatly increased. May we not then suppose that the liver under these circumstances breaks up such of the albumen as has been rendered unfit for nutrition, and that the excess of sulphocyanide represents this increased destruction of this altered material? After a certain period, the duration of which varies in different diseases, the amount of albumen in the blood becomes insufficient for the requirements of nutrition, and then we find, as we have before stated, a diminution in the quantity of the sulphocyanide in the saliva.

I have stated the conclusions which seem to me to arise from the above-mentioned facts only as *probable*, because the chances of errors of observation are so much greater when only a "colour test" is employed than when the amount of a substance is quantatitively determined. The labour involved, however, in employing a more accurate method would have been so great, and the number of cases collected so few, that it seemed to me to be advisable to attempt to obtain in the first place only a general view of the subject, and to leave the settlement of any questions that might arise to future and more careful researches.

I have, I trust, been able to prove that the presence of the sulphocyanide in the saliva is not due to accidental circumstances, but is regulated, as is the case with the secretions of other glands, by the operation of general laws; and above all, that the whole subject, whether viewed in the light of its scientific interest or its practical importance, is worthy of more attention than has been hitherto bestowed upon it.

A SUCCESSFUL CASE

OF

LUMBAR COLECTOMY,

OR

EXCISION OF A STRICTURE OF THE DESCENDING COLON

THROUGH AN INCISION MADE FOR A LEFT LUMBAR COLOTOMY.

WITH REMARKS.

BY

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On September the 8th, 1881, I was called to visit Mrs. D—, a widow, æt. 50, residing upon the coast of Suffolk, who was supposed to be dying from some unknown bowel trouble. She had had thirteen children, and without being an invalid had never been strong, and had never had any serious illness. For some years, however, she had been subject to constipation, for which she had taken, rather freely, mineral purgative waters. She had once, three years ago, gone three weeks without any action of the bowels. Her present illness began, as kindly

reported to me by Mrs. Garrett Anderson, about the middle of July, 1881, after a journey to London. The constipation became complete and was attended with considerable abdominal pain, coming on in paroxysms, but no fever. These symptoms continued for the two weeks she was in London, and during this time there was not only no action of the bowels but no passage of flatus, mucus, pus, or blood.

When she returned home she consulted her local medical attendant, who prescribed calomel internally and turpentine enemata, but without benefit. At this time vomiting appeared and lasted off and on for a week, accompanied with paroxysmal abdominal pain. Still the bowels were obstinate, no action taking place, and no passage of flatus. The patient became very weak from want of food and refused nutrient enemata.

On August 9th she passed into the hands of Mrs. Garrett Anderson for treatment. At this time Mrs. D— had had no action of the bowels for three weeks, sickness was frequent, and at times the vomit had a yellow fæcal appearance. Colicky abdominal pain came on at times and the abdomen was much distended. There was neither fever nor marked abdominal tenderness. The rectum on examination was found empty and flaccid, and no stricture could be felt. Enemata were given, but only about half a pint could be thrown up, and this was not retained, and always returned as it was injected.

During the next four weeks three very small evacuations were obtained. Sickness was only occasionally present and not severe. There was no fever and not much colicky pain. The abdomen became very large, coils of intestines being often visible. The patient was fed by small nutritive enemata. The urine was very scanty, loaded with lithates, and contained some bile and albumen. The patient had been treated with belladonna.

With this report I saw the patient on September 8th. She was in bed with a painfully emaciated and distressed

countenance and feeble pulse. Her abdomen was immensely distended, and through the thin abdominal parietes large coils of inflated bowels could be distinctly seen in which peristalsis was very visible. No one coil seemed tighter than another. No tumour could be felt. On examining the rectum the bowel was found empty and free from secretion or even a fæcal odour. No disease could be made out by the finger. The patient was unable to take the least nourishment, everything being immediately rejected.

The diagnosis was not difficult. It was clearly one of organic stricture, although the exact position of the stricture was not so evident. All probabilities, however, pointed to it being in the large intestines and probably in the descending colon. And the fact that the left loin was as full as the right encouraged this view.

With this diagnosis the treatment was not doubtful, for it was clear that relief could only be looked for by the operation of colotomy, since eight weeks at least had passed without any discharge that could be called relief to the bowel having taken place, and without even the passage of flatus. The feeble condition of the patient was, however, unfavorable for prognosis.

Consent for the operation having been obtained, arrangements were made for its performance on September 10th, when it was undertaken. Mr. Clover kindly gave the anæsthetic, and Dr. Collins, of Saxmundham, and Mrs. Garrett Anderson, assisted.

I started with the intention of performing a left lumbar colotomy, and for that purpose made my usual oblique incision midway between the ribs and crest of the ilium, with its centre corresponding to the outer border of the quadratus lumborum muscle, one inch behind a vertical line drawn from the centre of the crest of the ilium. The bowel was exposed without difficulty, and a hard indurated mass of new growth was then felt at the fore part of the wound, attached to what was supposed to be the descending colon passing downwards from the loin to the pelvis.

I opened the bowel and stitched it to the integument with six sutures, under the conviction from its relative position to the diseased mass that I was above the disease. No motion or wind, however, passed through the opening, and the finger after its introduction through the artificial opening into the bowel returned unsoiled. A closer examination was consequently made, and in separating the edges of the artificial anus, a sudden change in the position of the parts took place and there appeared through the artificial anus a growth which looked more like the neck of a uterus with a dilated os than anything else. Into this I passed a catheter, when wind and fæces at once escaped, proving to demonstration that the opening into the bowel which I had made was below the disease, although I had opened what from its position appeared to have been the upper part of the bowel. The artificial anus I had made was then at once unstitched and the true position of the parts carefully examined, and finding on manipulation that the strictured bowel was free and movable, that the stricture was an annular one, and that it did not involve more than one inch of the bowel, I determined to resect it and to fasten the divided ends of the bowel to the lumbar wound, and this I subsequently executed without much difficulty.

I commenced the operation by drawing the diseased part of the bowel well out of the wound and giving it in charge of Dr. Collins to hold. I then opened the distended upper bowel transversely about one inch and a half above the seat of the stricture, stitched the anterior border of its walls to the upper lip of the skin wound with three sutures, and allowed the bowel gradually to empty itself of quarts of slate-coloured liquid fæces. I took care, also, during this fæcal drain, to keep the anterior and lower surfaces of the wound clean. When the fæcal discharge had ceased or nearly so, and the parts had been cleansed, the section of the remaining two thirds of the calibre of the bowel was made; and I did this leisurely and carefully with a pair of scissors, by half-inch cuts

through the bowel, twisting bleeding vessels as they were divided and stitching each portion of the bowel as cut to the lips of the lumbar skin wound, carefully sponging the wound as I progressed. Indeed, it was by this same gradual process of dividing, sponging and stitching, that I separated the strictured segment of bowel from all its attachments (*see* Plate V, fig. 1), and so shut out the peritoneal cavity and completed the operation, and I may add that beyond the necessity of care I found but little difficulty in its performance. The upper orifice of the lower end of the bowel I secured at the lower lip of the wound in front of the artificial anus and in close contact with it (*see* Plate V, fig. 2). I did this with the view of facilitating the restoration of the continuity of the intestinal tract and closure of the artificial anus at some future time should the patient feel disposed to submit to the operation and the measure appear desirable, which I am disposed to question.

The case, subsequent to the operation, may be said to have done well, for a slow but steady convalescence followed and the patient on March 7th herself wrote that she was "miraculously well." I give the daily report in the words of my friend Dr. Collins, who watched the case for me with much skill:

"The operation was at three in the afternoon.

"September 10th, seven hours after operation.—Recovered well from shock of operation, but suffered considerable pain in wound. Took opium gr. j.

"11th (1st day, 11 a.m.).—Temp. 99·2°, pulse 113, resp. 30. Took three tumblers of milk and brandy during the night and slept a good deal at intervals.

"12th (2nd day, 1.30 p.m.).—Temp. 101°, pulse 116, resp. 34. No signs of peritonitis or secondary hæmorrhage. Doing fairly well. Two pints of urine drawn off at four different times. It was slightly albuminous. 10.25 p.m.—Temp. 101°, pulse 110, resp. 32. Wound looking well.

"13th (3rd day, 12.15 p.m.).—Temp. 102·6°, pulse 118,

resp. 30. 10.20 p.m.—Temp. 102·4°, pulse 112, resp. 28. Patient very weak and exhausted, bowels having acted continually since morning, the dressing being changed fourteen times. Wound looking rather dusky. Dressed only with oakum. Ordered chalk and opium mixture every four hours. I may here mention that the patient is very unmanageable, it being quite a battle to make her take either medicine or food of any description. I never met such a woman in all my life.

"14th (4th day, 12 a.m.).—Temp. 101°. Bowels only acted slightly since last night. Wound looks much better and healthier. 10.30 p.m.—Temp. 103°, pulse 104. Bowels again very loose and slimy, and the patient very weak, taking hardly two tumblers of milk and brandy in the twenty-four hours. Very intractable. Wound looking rather red at edges.

"15th (5th day, 12.15 p.m.).—Temp. 102·4°, pulse 110. Wound looking rather ashy. I removed the two superficial stitches. The wound gaped considerably and was evidently going to slough. Dressed it with lint soaked in solution of carbolic oil, 1 in 20. 11 p.m.—Temp. 101·2°, pulse 109. Wound washed out with strong solution of carbolic acid and a charcoal and linseed poultice applied, and ordered to be changed every four hours at least. Opium gr. j 4tis horis.

"16th (6th day).—Pulse 104. Some sloughs separating at outer end of wound. Half a pint of milk with 1½ tablespoonful of brandy was all the nourishment that could be got down. The opium could only be got down at irregular intervals and with great trouble. Mouth becoming aphthous and pulse very weak. 6 p.m.—Temp. 99·4°, pulse 110. Sloughs separating. Still real difficulty in getting down nourishment.

"17th (7th day, 2 p.m.).—Temp. 99·4°, pulse 110. Wound more healthy, washed well with solution of iodine and water. One drachm of Tinct. Iodi rubbed up with each poultice. 10.50 p.m.—Temp. 98·6°, pulse 100. Had great pain in wound. Sloughs were separating well,

so discontinued iodine. Patient weak and taking little nourishment. The mouth and throat have been painted frequently with mixture of borax and glycerine.

"18th (8th day, 12.30 p.m.).—Temp. 98·8°, pulse 108. Mouth better, taken more nourishment and enjoyed it. Wound cleaning. The orifice of the artificial anus seems very deeply placed. Wound dressed with lint soaked in 1 in 20 solution of carbolic oil and poulticed as before. 11 p.m.—Temp. 100°, pulse 104. Pulse feeble. Wound indolent looking; dressed as before.

"19th (9th day, 12 noon).—Temp. 99·4°, pulse 100. Pulse stronger. Taken one drachm of brandy every hour and sixteen ounces of milk since last night. Ordered Pil. Quin. Sulph. gr. ij every two hours. 11 p.m.—Temp. 99°, pulse 104. Wound looking more healthy. Has taken half a cup of beef tea twice to-day besides milk and brandy.

"20th (10th day, noon).—Temp. 98·4°, pulse 100 and stronger. She has taken more nourishment. The wound is looking healthier and healing up rapidly from bottom. Still dressed with lint and carbolic oil.

"21st (11th day, 1 p.m.).—Temp. 98·8°, pulse 100. Had diarrhœa all the night, stopped by chalk and opium. Pulse fairly strong. Wound still healing. Nourishment taken better. Taken ten quinine pills during the last thirty-six hours with only slight headache. Pills only to be given now every six hours.

"22nd (12th day, 3.30 p.m.).—Temp. 98°, pulse 104. Feeling better. Wound healing well; dressed with lint and carbolic oil 1 in 30.

"23rd (13th day, 5.15 p.m.).—Temp. 19°, pulse 100. Quinine pills discontinued since mid-day yesterday as patient refused to go on with them.

"24th (14th day).—Temp. 98·6°, pulse 101. Wound only half depth and width. Fæces pass out freely from artificial anus and without pain.

"25th (15th day).—Temp. 98·6°, pulse 104. Wound healing well.

"26th (16th day).—Temp. 99·4°, pulse 108. Wound still healing up rapidly, but patient only takes liquid food, brandy and milk, and a very small quantity of ox-tail soup. Considerable discharge of sweet pus from a small passage running along the outer side of the lower bowel. A probe passed about two inches into the small passage and a small strip of lint soaked in carbolic oil kept in opening.

"27th (17th day).—Temp. 98·8, pulse 110. Going on well. Still a considerable discharge of pus from the opening mentioned.

"28th (18th day).—Temp. 98·8°, pulse 110. Going on the same. Wound healing but looking pale. Patient has taken some baked custard the last day or two. Throat still relaxed. Has been at last induced to use an astringent gargle which has been made up for the last week.

"29th (19th day).—Temp. 98·8°, pulse 112. Patient much stronger. Pus discharge much less. Has taken much more nourishment and the wound looks consequently a better colour. Some solid food has been taken, the gargle having strengthened the throat. Wound healing kindly, is now only about one third the size; dressed with red lotion. The patient would not knowingly take iron in any form, so ordered Quin. Sulph, Zinci. Sulph., Ferri Sulph., āā gr. ss. in a pill to be taken three times a day.

"October 19th (39th day).—The artificial anus well established. The wound has cicatrised, and fæces pass freely through it. Her appetite has been very good, and patient is up daily, and has been growing quite fat, and says she feels better than she has for more than a year. Frequent enemata have been given by the natural anus, and a teacupful of fæces, like marbles, has been brought away per anum. These must have been impacted in the lower end of bowel for some time.

"31st.—Patient has been going on well. Enemata administered per anum passes through lumbar opening,

proving its present patency. Artificial anus is well formed.

"November 10th.—Patient is going on most satisfactorily.

"December 2nd.—Patient continues to go on well, and slowly regains strength. She looks very well indeed in the face, and eats, drinks, and sleeps well; she goes into the drawing-room daily. About every four or five days there is some discomfort in the rectum, and an enema brings away about half a dozen pieces of fæces as big as half marbles. The bowels are well relieved by the artificial anus, and the motions are always solid.

"No persuasion will induce our patient to take any tonic."

Remarks.—I have brought this case before the Fellows of this Society to introduce to their notice an operation which I believe to be applicable to certain forms of stricture of the descending colon; and since the patient upon whom it was performed is now well, her disease having been removed, I trust I shall not be considered injudicious when I urge its adoption.

The case before you is the only one in which I have attempted the operation I have described, though I may say that the idea of removing an organic stricture of the large bowel through the wound made for a left lumbar colotomy suggested itself to me several years ago, after having seen, both in operations of colotomy as well as in the post-mortem room, many examples of annular or localised stricture of the bowel which were freely movable in the peritoneal cavity, free from all attachments, and within easy reach of the surgeon's fingers through the lumbar wound. For it is in these cases, and in these alone, that the operation is possible.

In what proportion of cases of stricture of the descending colon this operation is applicable, it, possibly, may be difficult to decide, but since pathological inquiries tell us in very decided language (*vide* Table A.) that in chronic intestinal obstruction the seat of stricture in three out of

TABLE A.—*Causes of Intestinal Obstruction, excluding Hernia.*

Being an analysis of 124 consecutive cases extracted from the post-mortem records of Guy's Hospital, by Dr. Hilton Fagge, from 1854 to 1868 ('Guy's Rep.,' 1868); and Mr. Russell, from 1868 to 1876 (unpublished).

Guy's cases.		
33	ACUTE OBSTRUCTION	1 Internal hernia. 7 Twists (volvulus). 25 Bands. { 14 Lymph. 6 Diverticula. 2 Appendicæci. 2 to neck of hernial sac. 1 from pedicle of ovarian tumour.
76	CHRONIC OBSTRUCTION	3 Fæcal impaction. 3 Mechanical pressure of tumours. 47 Stricture { 2 Small intestine 45 Large 23 Matting together of intestinal coils from peritoneal and cancerous disease. (Contractions.)
15	INTUSSUSCEPTIONS	2 Rectal. 7 Ileo-cæcal. 6 Small intestine.
124		

Analysis of 63 cases of stricture of intestines as given in a paper by Coupland and Morris. ('Brit. Med. Journ.,' Jan. 26, 1878.)

	Middlesex cases. 28 large intestines, 3 small.		Pathological Society. 31 large intestines, 1 small.		General Total.
33 Rectum and sigmoid flexure	24	...	21	=	78
7 Transverse colon with hepatic and splenic flexures	3	...	9	=	19
5 Cæcum or ileo-cæcal	1	...	1	=	7
					104

every four cases is located in the descending colon, and that about one third of these cases is of an annular or local character, I am disposed to think that the instances in which this operation may have to be considered are not few.

It should also be remembered that of all strictures of the large intestines other than those clearly due to inflammatory or syphilitic causes, the annular approaches in its histological as well as clinical features the simple growths; and that whilst, on the one hand, the histologist describes them as being composed of epithelial, cylindrical, or adenoid elements, the pathologist will demonstrate the hard clinical and pathological fact that such growths are rarely attended with any metastatic or other disease, and are for the most part local.

Under these circumstances I submit that the expediency of removing the strictured bowel, where practicable, is more than demonstrated, and I think I may add that the method of doing so by the operation I have brought under your notice will prove to be the best means.

Should this opinion be accepted, some change in practice may be required, for it would be wise to entertain the operation of excision of the stricture at an earlier period of its progress than it has hitherto been the custom for physicians or the majority of surgeons to entertain that of colotomy. Since the operation of excision of the stricture would be more readily performed when the bowel above the stricture was undistended and comparatively healthy than when it was full of retained fæces and probably ulcerated from over distension. The operation, moreover, when performed under these more favorable circumstances, would be safer, since with healthy bowel above and below the strictured segment the surgeon may with more confidence draw the diseased portion upwards from the pelvis or downwards and backwards from the splenic region, and consequently remove it with greater safety and facility.

The consideration of the operation of excision in any

given case will consequently have an important bearing upon that of colotomy, and give a help to that operation where it is sadly needed. For it will encourage physicians and surgeons to entertain the question of operative relief by colotomy or excision (colectomy) as soon as the diagnosis of organic stricture of the descending colon has been made, and not to postpone the consideration of the one or other operation till, as I have heard it advised, obstruction has existed for six weeks, when the chances of success are small.

In the case I have recorded it is true the chances of obtaining a good result even by colotomy were very remote, since it had been allowed to drift eight weeks with practically complete obstruction until the powers of the patient were almost exhausted, and it was probable that some secondary changes had taken place in the cæcum or distended colon.

I consequently advance the case rather as a warning than as an example, and to show how, even under the most unfavorable circumstances, the operation of excision (colectomy) or colotomy may prove successful.

I would therefore ask my medical as well as surgical friends to accept this operation of excision of a localised stricture of the descending colon as an additional means of giving relief and possibly of curing a certain proportion of cases of stricture of the bowel. To consider it in all cases of stricture of the descending bowel not rectal, and to do so as soon as the diagnosis of mechanical obstruction has been made, and before the symptoms of obstruction are a source of anxiety or of a threatening character.

I would ask the surgeons to examine every case in which the operation of left lumbar colotomy is entertained, with the view of determining the feasibility of excising the stricture before establishing an artificial anus. And I would suggest that the different steps of the operation should be carried out in the way I have described.

I cannot regard the operation I have performed in a much more serious light than I do a colotomy in which

the peritoneum has been injured. Whereas the prognosis in the two cases varies greatly, for after the operation of excision the hopes of a cure may with reason be entertained, whereas after a colotomy performed under the same conditions the operation can but postpone the evil day and not avert it. The operation of colectomy being curative and that of colotomy palliative.

Dr. Goodhart has kindly made a careful examination of the preparation of resected bowel and reports as follows:

"The part sent to me for examination is the whole circumference of the bowel with some of the surrounding subperitoneal fat. One half of it measured lengthwise is puckered and indurated, forming a hard, nodulated mass an inch or so in length. One of the free ends of the specimen shows the mucous membrane thickened and granular looking, and the submucous and muscular coats converted into tough fibrous-looking material. The bowel being tightly strictured at this part.

"A vertical section of the tissues concerned in the stricture was made and examined microscopically and shows appearances such as are depicted in the two accompanying drawings (*see* Plate VI, figs. 1 and 2) from the pencil of Miss Alice Boole. Alveoli are spread through a predominating fibroid tissue, and these are lined or filled with epithelial cells, many of them sufficiently columnar to allow the growth to be called a columnar epithelioma, but many of them also of a somewhat less specialised type, such as are found in many cancers. The amount of fibrous tissue is, however, the dominant feature of the growth.

"The disease is by no means an uncommon one. I am not prepared to state the numerical proportions of such to a more diffused and ulcerating form of cancer of the sigmoid flexure, but this I know that it is a common experience to find obstruction of the sigmoid flexure which to the naked eye is strictly alveolar and puckered like this, giving the appearance of a ligature tied round the bowel, and which cannot be said to be cancerous from the coarser features of the disease. It is equally

certain that these cases, when examined microscopically, prove to be cancerous, and they generally show similar features to those depicted from this case.

"I should, therefore, be quite prepared to go with the author of the paper in insisting that there is a class of cases, and not a small one, in which the entire disease might be removed, so far as the extent of the disease is concerned,—if surgery should decide that the operation is feasible and safe.

"I may add that from what I have seen in the dead-house, I believe that the operation would be feasible in not a few cases, though in the case of females the frequent adhesion of the puckered part to the ovary would have to be borne in mind. And I believe, too, that the existence of such a condition can be found occasionally by the absence of diarrhœa or rectal discharge, by the absence of any symptoms save troublesome constipation until the supervention of *complete obstruction*, which comes about suddenly by a twist and falling over of the distended bowel above the stricture, as in this case, rather than by the *stricture* itself."

Note—On August 4th I saw, with Dr. Collins, Mrs. D—, who had been suffering for some weeks from symptoms of steadily increasing obstruction which were due to a rapid closing of the artificial anus. This I dilated with my little and index fingers, and subsequently with a short conical bougie half an inch in diameter at its apex and one inch at its base. At the same time some lumps were felt in the abdomen, which were probably fæcal. Since this dilatation was effected, I have learnt that good motions have passed and that Mrs. D— is relieved.

DESCRIPTION OF PLATES V AND VI.

Stricture of the descending Colon. Portions removed through incision for a Left Lumbar Colotomy (THOMAS BRYANT, F.R.C.S.).

PLATE V.

Fig. 1.—Portion of descending colon. (*a*) Lower orifice of stricture.

Fig. 2.—(*a*) Upper end of lower bowel before its closure by sutures; (*b*) artificial anus.

PLATE VI.

Fig. 1.—Columnar epithelioma, microscopical section. Hartnack, obj. 3, ocu. 3.

Fig. 2.—Ditto, ditto. Hartnack, obj. 3, ocu. 4.

Fig. 1.

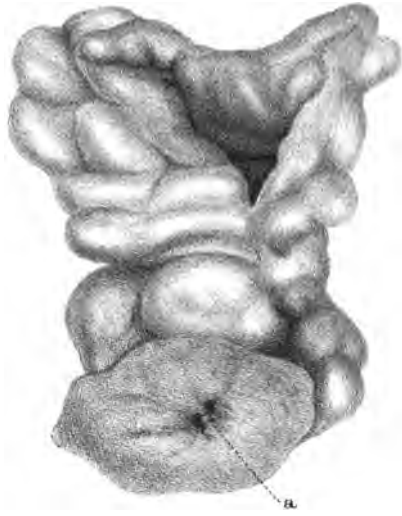


Fig. 2.

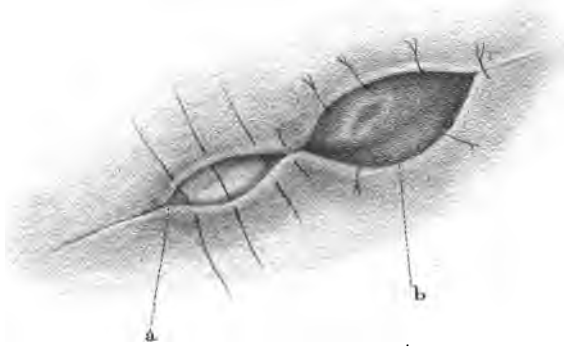
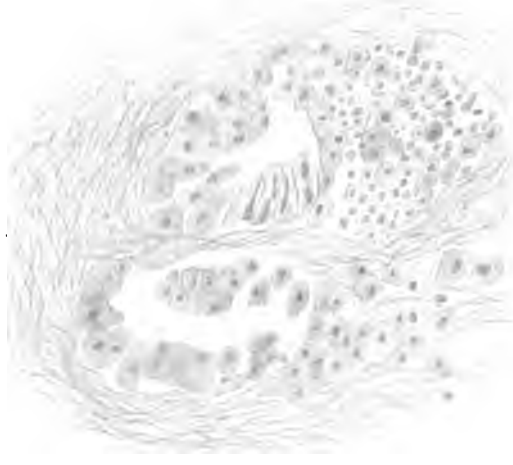


Fig. 1.

Fig. 2.



ON A CASE

OF

TUMOUR OF THE BLADDER (IN THE MALE)

SUCCESSFULLY REMOVED THROUGH A PERINEAL SECTION OF THE URETHRA;

WITH REMARKS ON THE APPLICABILITY OF THE OPERATION IN CERTAIN CASES.

BY

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Thomas Reading, æt. 29, consulted me July 26th, 1880. I learned that eight years previously he had passed "a piece of gravel the size of a pea." After this he felt nothing unusual until three years ago, when his micturition became more frequent, and was followed by pain in the end of the penis, also occasionally blood appeared in the urine, especially after exercise.

With these symptoms, I sounded the patient, and I found a small calculus, which on August 5th was easily crushed and removed. It was composed of oxalate of lime.

Very little improvement followed the operation; the

bladder was not quite emptied by the natural efforts; the gum catheter was used daily, and on two occasions gave signs of the presence of something in the bladder, which a subsequent exploration of the lithotrite did not discover. Such results were unusual and somewhat puzzling. Being relieved, he resumed his employment, and was occasionally seen relative to the still existing slight symptoms, which, however, gradually increased.

On October 5th I examined the bladder, and removed a quantity of phosphatic deposit with the lithotrite. I then seized what at first felt like calculus, and partially crushed under pressure; but it was evidently fixed, giving me the impression that I was dealing with a portion of stone partially impacted, and that the remainder would be beyond my reach. More phosphatic matter being washed out, I decided to watch the result for a short time, and to open the bladder if necessary, as I have before done in cases of sacculated calculus. Accordingly, as after three weeks he had received very little benefit from the last operation, I decided to cut as in median lithotomy, and did so on the 6th of November, 1880.

Having introduced my finger well into the bladder, and pressure being made above the pubes, I soon recognised a tumour, about the size of a chestnut, growing apparently from the opposite wall or fundus, and somewhat to the patient's left, coated with phosphatic matter, and evidently the fixed body I had formerly seized with the lithotrite and denuded of its sabulous covering. Taking a pair of small forceps, I adjusted them to a full and firm hold, and then twisted off the mass without difficulty; a small piece or two were subsequently withdrawn, but the tumour appeared to be entirely removed, and very little bleeding followed. He had no bad symptoms, made a rapid recovery, speedily regained good health, never having had any return of symptoms since the operation, now about fifteen months ago.

During that period I have seen him only two or three times so as to be assured of his continued health. He is

here to-night, and I should scarcely know him to be the same individual I first saw a year and a half ago, so greatly has he gained in flesh and strength.

Taking the excellent result of this case in connection with those described by Professor Murray Humphry in 1879, recorded in the 62nd volume of our 'Transactions,' the case of Billroth cited in the same paper, with another recently under the care of Mr. Davies-Colley, related by him at the Clinical Society in 1880,[1] all of them males, together with an example of tumour in the bladder of a female brought before this Society by Mr. Berkeley Hill during the present session, it is impossible not to arrive at an important conclusion respecting them. With such evidence, I think it will generally be conceded that certain tumours of the bladder are met with, not numerous it is true, although probably not so rare as they are generally supposed to be, which are capable of being successfully removed by surgical operation, but which are invariably fatal if left to follow a natural course. I do not hesitate to say, on reviewing my experience, that I have met with several cases apparently of disease of the bladder, but presenting obstinate and perplexing phenomena, with gradual tendency to grow worse in spite of treatment, for which I should now advise a careful exploration by perineal incision.

I have long known that it is not possible by any sounding, however delicate, to diagnose with certainty during life the presence of villous growth, epitheliomatous formation, or soft fibrous tumour, which have been subsequently discovered by post-mortem examination. I refer to examples of all these in which such investigations have been carefully made by myself during life, and subsequently after death. And this fact I have emphatically stated in discussing the subject on more than one occasion in my written works. The presence of cancerous growths, when fairly developed, can, on the other hand, be ascertained during life without much difficulty. But the

[1] 'Trans. Clin. Soc.,' vol. xiv.

tumours before referred to always yield *débris* from time to time, both naturally and by washing out the bladder, and from microscopic examination of these specimens some light on their nature can be gained. I was much impressed by the autopsy of a patient whom I saw from time to time at considerable intervals during a few years, and who suffered severely, especially during the latter months of his life, about two or three years ago. On examining the bladder after death I found a tumour, as large as a moderate-sized apple, attached by a pedicle not a quarter of an inch in diameter. I regretted exceedingly that I had not opened his bladder and removed the tumour, which could have been done without any difficulty. The mass, although large, was very soft, and it was doubtless owing to this fact that I did not detect its presence as a tumour by examination during life. He passed small masses of villous growth, but thinking he was probably the subject of numerous small sessile flocculent growths of that nature, a common form, I never hoped to gain anything by opening the bladder.

I should no longer hesitate, in presence of a group of symptoms which to my mind suffice to suggest strongly the presence of such tumour, to advise exploration by perineal incision, for these growths invariably lead to a fatal issue sooner or later. Their course is sometimes very slow, but when it is rapid, and also in the later stages of their development, they produce extreme suffering. On the other hand, the operation advised is not a severe one.

The mere opening of the urethra, short of the neck of the bladder, by incisions in the line either of median or lateral lithotomy, does not involve a dangerous wound; the risks of lithotomy being occasioned much more by injury inflicted on the bladder itself in removing the stone than by the preliminary incisions made by the knife.

Perhaps of the two methods the median incision inflicts less mischief, and involves less risk than the lateral. For the purpose now under consideration the section should be strictly limited to structures external to the neck of the

bladder, which is barely to be approached with the knife. Such an operation amply suffices for exploration, as it would also, probably in four cases out of five, for removing any tumour discovered, although one of exceptional size might demand for its extraction a suprapubic operation, as in the case by Billroth.

The symptoms I have referred to as rendering an exploration of the bladder by perineal incision worthy of consideration in any given cases, may be thus sketched:

The patient, who is usually young or at middle age, is subject to attacks of hæmaturia, which are mostly slight at first, often with long intervals of freedom from bleeding. During the earlier stage it sometimes happens that no other sign of deranged urinary function is present besides the hæmaturia. It is to be observed that in the act of micturition the stream of urine may at its commencement be natural in colour and unstained, but that it may finish with a tint of bright red, a significant and important phenomenon in relation to diagnosis. In hæmaturia from the kidney the mixture of blood and urine is intimate and uniform throughout, and the mixture almost invariably presents a duller tint. The stage thus indicated may last from one or two to seven or eight years. Then, the act of micturition becomes more frequent and sometimes painful; these characters appear irregularly, and by no means uniformly in amount and degree.

The pain is not necessarily aggravated, as in stone in the bladder, by active movements of the body. In later stages the pain is not necessarily severe, except from obstruction to the outflow of urine by clots. In cancerous disease pain is much more distressing; at the same time indurated masses may be detected by either rectal or vesical examination, or by both. Free hæmorrhage, again, is not an evidence of the presence of calculus, but the contrary, the characteristic bleeding of calculus being slight, and directly corresponding to the amount of movement permitted to the body.

As the tumour progresses hæmaturia is more frequent

and more persisting, and blood is always to be found in the urine under the microscope.

Urethral obstruction causing retention sometimes takes place from the expulsion of portions of organised tissue, sometimes by the flocculent extension of a villous tumour, situated close to the neck of the bladder, being carried into it by the current, as I have seen on one occasion. Retention of urine more or less complete, with much suffering, may appear in the latter stages; while the patient loses flesh and treatment of any kind is ineffective.

With a case presenting such symptoms, the explanation of which appears not to be ascertainable except by the hypothesis of a growth, I think the question of making the opening described should be entertained. If no valid objection exists to the proceeding, it should be undertaken and the bladder should be explored. If after careful examination no tumour or other manifestly abnormal cause of symptoms is found, it will, I think, generally prove serviceable to adopt the following course:—A large india-rubber catheter should be introduced by the wound into the bladder and allowed to remain there, in order to withdraw all the urine by that route, perhaps for some days. This process is sometimes useful in chronic disease of the bladder by permitting the expulsive apparatus and the urethra to remain at rest for a period of time, by which means a healthier condition may be acquired, and after this the wound may be permitted gradually to close. If no improvement has taken place, it is at least almost certain that no injury can subsequently arise from the operation.

Mr. Stanley Boyd, lately Surgical Registrar of University College Hospital, has been good enough to examine a portion of the tumour from this case, and his report thereon, which is as follows, will conclude this paper.

"A small piece, taken from the surface of the tumour, was handed to me for examination. It was thickly encrusted with phosphates, and beneath these the surface was finely irregular. On section, the growth was firm and of uniform

consistence; but its structure could only be guessed at as fibrous.

"Microscopically, it consisted of fine bundles of fibrous tissue, having a general direction vertical to the surface. Small round cells were scattered pretty copiously at parts, especially towards the free surface, but there was no regularity in their distribution. The tumour is therefore a simple fibroma.

"A knowledge of its structure could be obtained only by the examination of a considerable number of sections; for in many parts the structure was very indefinite, and was rendered still more so by a cloud of fine granules which cleared up on the addition of acetic acid.

ON

WOUNDS OF THE THECA VERTEBRALIS

WITH

DISCHARGE OF CEREBRO-SPINAL FLUID.

BY

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(Received March 6th—Read April 25th, 1882.)

In a communication to the Society, published in the sixtieth volume of the 'Transactions,' I related the case of a boy, æt. 13, who received a stab in the loins, and whose case presented the peculiarity of a copious discharge of watery fluid from the wound. This fluid must have been either urine from the ureter or cerebro-spinal fluid, and it was difficult to decide which. I argued in that paper that it was probably from the ureter, and I proved, by experiment on the dead subject, the ease with which the ureter could be reached by a stab wound in that situation. The enormous quantity, and the copious and continuous flow of the fluid, were also features in the case which were difficult of explanation on the hypothesis that the fluid was cerebro-spinal; while, on the other

hand, its composition was at least very similar to that fluid, and no urinous constituents could be clearly recognised in it by chemical examination.

I will now proceed to relate an undoubted case of wound of the spinal theca which occurred recently under the care of my colleague, Mr. Rouse, at St. George's Hospital, and will then subjoin notes of a similar case which was published a few years ago in one of the journals, and which proved fatal, and was examined after death.

For permission to use the notes of the subjoined case (which were taken by Mr. Fuller, the house surgeon) I am indebted to Mr. Rouse.

William W—, a schoolboy, æt. 11¼, was admitted under Mr. Rouse's care suffering from a small punctured wound of the back June 25th, 1881.

History.—On June 19th he was stabbed by another boy whilst at play with a penknife having a blade about one inch and three quarters in length. There was trivial bleeding at the time of the accident, but shortly after he was taken home it was noticed that "watery stuff" ran from the wound.

The flow ceased on June 22nd, the wound having scabbed over. On the evening of June 24th the patient rubbed off the scab, the "watery flow" recommenced, and continued till the time of admission.

Between June 19th and June 25th he was under medical treatment at home. He was kept in the prone position, and water dressing applied to the wound.

On admission (June 25th).—A pale, delicate-looking lad. No head symptoms of any kind. No spinal symptoms. No loss of muscular power or sensation. Gluteal reflex and spermatic reflex unimpaired. Trousseau's tâche cerebrale easily elicited. Bowels open and acting regularly. Urine passed easily; reaction acid; characters normal.

N.B.—The boy was carried into hospital in the prone position. He refused to stand up, and his friends evidently wished him not to attempt to do so; and the point was not

pressed. A careful examination in bed showed this was *not* due to loss of muscular power.

A wound exists in the small of the back. Draw a line round the body on a level with the highest point of the crests of the ilia, one inch and a half below this line, and a quarter of an inch to the right of the median line, is a small vertical wound just a quarter of an inch long.

There is no redness around the wound, and no tenderness. Pressure about the wound caused no fluid to exude, but, on making the patient wriggle about, a copious flow of limpid, colourless fluid takes place, so that in half an hour almost a test tube full was collected.

A piece of plaster was placed over the wound, the whole sealed with collodion. Prone position maintained.

26th.—Slept well. Tongue clear. Appetite good. Fluid having escaped under the collodion, the house surgeon drew the edges of the wound together with two fine silk sutures; over this a minute piece of lint was placed, over this plaster, and the whole sealed with collodion.

29th.—No further escape of fluid. Prone position maintained.

July 3rd.—Slight irritation from collodion. Prone position maintained. Health excellent.

9th.—Lint and collodion came off. Wound healed; slight bulging at seat of wound; sutures removed.

14th.—Bulging increased since last note. Dressed yesterday and walked about ward: no symptoms have resulted.

24th.—Boy, kept till now for observation, is perfectly well. All movements natural; sensation perfect. A simple scar remains. Discharged.

October 25th.—Seen by house surgeon at 5 U, Peabody Avenue, Pimlico. A small, slightly depressed, pink cicatrix, the size of a barley-corn, remains. Boy seems in perfect health though looking delicate. All movements natural.

Characters of fluid.—Clear, watery, saline to taste, re-

action alkaline; a very faint trace of albumen (?). Argentic nitrate throws down a white precipitate.

Mr. Donkin reports on the fluid that it has a "specific gravity 1·008, and contains altogether 1·12 per cent. of solid matter dissolved and suspended. Of this 0·22 is organic, the residue blackening on heating and burning partially away, leaving 0·9 per cent of inorganic salts. These consist chiefly of chloride of sodium and carbonate of sodium in about equal proportions (the latter no doubt formed, partially at least, on incineration), with traces of phosphates. The original fluid does not change (apparently) on heating, and does not reduce Fehling's solution."

The fluid above described was collected between 4.30 and 5 p.m., June 25th. No doubt some of the organic material above mentioned was due to the test-tube scraping off epithelial scales whilst the fluid was being collected. A few epithelial cells were found under the microscope.

The only case I have found of punctured wound of the spinal membranes in which a dissection was obtained, is recorded in the 'Lancet,' vol. ii, 1876, p. 457, by Surgeon-Major Gribbon. The patient, a man æt. 32, stabbed himself in the back of the neck in a fit of insanity, and died nineteen days afterwards. The wound, which opened the spinal theca, was inflicted with a penknife. It was ¾ inch in length, and a probe passed down it impinged on the arch of one of the vertebræ, apparently the second or third cervical. There was an oozing of a sanious fluid from it continuously and in considerable quantity. On the fourth day this fluid became quite clear, and on the fifth day some of it was collected for examination. It was limpid, of a pale straw colour, sp. gr. 1012, alkaline, and with a trace of albumen. There seemed to be no prominent symptom beyond pain in the neck, and nothing to indicate any lesion of the spinal cord. On the eleventh day, however, the pulse and temperature began to rise, and continued considerably above the normal till his death. It was not till the sixteenth day (three days before death)

that the inspirations are observed to be remarkably slow (twelve in a minute) and thoracic, "like a succession of long-drawn sighs." He was conscious up to the day before his death, and on that day when asked whether he felt pins and needles in his feet he said that he did. He had also complained of cramp and numbness in the right hand, but there seemed no satisfactory proof of any symptom pointing definitely to lesion of the spinal cord. Death was attributed to coma, the result of imperfect respiration. The oozing of fluid from the wound continued up to the last day of life. Its quantity depended partly on whether the wound was plugged or no, and partly on the movements of the patient. "After any considerable change of posture, such as to quicken the circulation, it welled out as from a spring. On one occasion, after it had been necessary to pass a probe into the wound and he had been supported up while this was done, it actually issued out *per saltum*, precisely as arterial blood would behave, and the movement was synchronous with the pulse." On the whole it was estimated that the loss of cerebro-spinal fluid averaged four to five ounces a day.

On post-mortem examination the dura mater corresponding to the second and third cervical vertebræ was of a darkened colour and softened. A probe introduced underneath at a point lower down and passed upwards, emerged readily through an opening, which was ill-defined in consequence of the disorganised condition of the membrane. The ligamenta subflava in this region were also highly vascular, and intimately attached to the dura mater by recent exudation. The arachnoid in the upper cervical region was injected, especially the parietal layer, and its prolongations encasing the posterior roots were thick and opaque. The vessels of the cord were here numerous and large, but its substance appeared healthy. There was no fluid in the subarachnoidean space. There was, however, some fluid in the ventricles of the brain. The brain substance is described as "hardly of natural firmness."

In this case it is rather difficult from the account of

the symptoms and post-mortem examination to make out the exact cause of death. The man was doubtless a lunatic, and the brain seemed somewhat softened; but that condition had apparently existed for some time, and the fatal result is distinctly referred by the surgeon in charge of the case to loss of function of the phrenic nerve, the origins of which were thought to be implicated in the inflammatory material poured out around the wound. Allowing this, we see that, apart from this merely incidental complication, the wound of the spinal theca, and the consequent loss of cerebro-spinal fluid, produced no symptoms whatever.

If, now, we compare the two cases just reported with the one which I related in the 60th volume of the 'Transaction,' we shall see so striking a resemblance as to justify a strong suspicion that that case also was one of wound of the theca vertebralis and not of the ureter. True, the flow was enormous, far greater than in Mr. Rouse's patient, and much greater than in Surgeon-Major Gribbon's case. But it seems natural that the flow should be more copious the lower down in the spinal canal the wound may be, whilst the greater quantity of fluid lost in any case as compared with Mr. Rouse's may have depended on a difference in the size or direction or freedom from obstruction of the wound. One symptom is common to all three cases, viz. the great increase in the flow which was produced by muscular movements.

These cases seem to me to have an important bearing on the physiology of the cerebro-spinal fluid, and on the consequences of its loss, for they go to show that its uses are purely mechanical, that it can be secreted (probably by mere filtration) in enormous quantity and with the greatest rapidity, and that its withdrawal, if not very sudden, is not accompanied by any symptoms whatever, so that Hirschfeld's assertion that "its loss in considerable quantities always causes death" is perfectly unfounded.

I am not quite so sure, however, that the sudden and total withdrawal of the cerebro-spinal fluid through a large

open wound would be equally harmless, though on this point our experience is not very considerable. The withdrawal of cerebro-spinal fluid in tapping a spina bifida does not seem ever to lead to any bad symptoms, but then the withdrawal is only partial and is counterbalanced by the pressure exercised on the tumour by the surgeon and by the atmosphere. Spina bifida tumours are, however, sometimes removed. Dr. Wilson, of Claycross, has related such a case in the 14th volume of the 'Path. Trans.,' and the operation was quite successful, and the same gentleman has, I know, operated on other cases successfully. But in the first case, at any rate, the tumour did not communicate with the subarachnoidean space. In a case of the sort which I operated on many years ago no immediate symptoms followed the incision of the tumour and evacuation of the cerebro-spinal fluid. But in an infant in whom I removed a tumour which turned out to be spinal, *i.e.* to have a small pedicle communicating with the spinal theca, as soon as the cerebro-spinal fluid escaped the child fell into a condition of somewhat alarming syncope, and my friend Mr. J. H. Morgan, who was assisting me, told me that he had witnessed a similar occurrence at the Hospital for Sick Children. We can easily understand that even if the uses of the fluid are merely mechanical, and if it can be re-secreted as rapidly as possible, yet its too sudden withdrawal may cause great interference with the functions of the nervous centres which it supports.

APPENDIX.

The following case, for which I am indebted to Sir J. Paget, and in which the discharge of spinal fluid was spontaneous, *i.e.* due to perforation of the theca in disease, is very interesting, as showing that even when the per-

foration is due to inflammatory ulceration, inflammation need not necessarily be propagated to the membranes of the cord. I give the case in Sir J. Paget's own words:

"A lady, about twenty years old, had carious ulceration of her sacrum, and after long suppuration was slowly wasting and slowly dying with hectic fever. One night there was a rapid flow of perfectly colourless and limpid fluid from one of the apertures of suppuration at the side of the sacrum. The sheets and blankets were widely wetted through; but, except for the discomfort, she would not have known that anything had happened. Similar fluid continued to escape during the remaining week or ten days of her life, but I could not find any reason to believe that the escape hastened her death or in any way affected her condition.

"In the first flow I think that there must have been from four to five ounces of fluid, on each of the following days about two ounces, but it was impossible to collect it. It was all so colourless, clear, and limpid that I could not have a doubt about its being spinal fluid."

TWO CASES OF LARYNGEAL GROWTHS,

IN ONE OF WHICH

NUMEROUS SESSILE, IN PART SUBGLOTTIC RECURRENT PAPILLOMATA,

AND IN THE OTHER

A LARGE, HARD, BROAD-BASED VASCULAR FIBROMA,

WERE SUCCESSFULLY REMOVED BY

ENDOLARYNGEAL, MAINLY GALVANO-CAUSTIC, OPERATIONS.

BY

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(Received March 14th—Read May 23rd, 1882.)

Endolaryngeal operations, unheard of twenty-two years ago, are now so frequently performed that the publication of individual cases is only justifiable if they offer special points of interest with regard either to the texture, size, and situation of the growths, or to the methods employed in removing them, or especially if the operations have been performed in cases generally believed to be unsuitable for endolaryngeal interference. All these points of interest are combined in the two cases which I have now the honour to bring before the Society.

CASE 1. *Multiple, sessile, in part subglottic papillomata; total aphonia; slight dyspnœa on exertion; repeated recurrence; final cure.*—Miss F. St., æt. 20, consulted me on April 4th, 1881, for complete aphonia, which had gradually developed from a slight hoarseness left after a cold caught twelve months previously. The voice had been entirely lost since the end of September, 1880. For some time past she had felt slight difficulty in breathing on exertion, and had a sensation as if some foreign body were in her throat, but there had never been either pain or dysphagia. Ulceration of the left vocal cord had been elsewhere supposed to be the cause of the symptoms present. There were no other complaints; and, in short, the only organ at fault was the larynx.

The laryngoscope showed that almost the entire glottic opening was occupied by, and its surroundings covered with, irregular, small, whitish, cauliflower-like masses. The whole anterior commissure was filled with growths, which extended thence over at least four fifths of both the vocal cords. The right vocal cord was completely embedded in these structures, and concealed from view. On the left side the growths covered only the upper surface of the vocal cord, and on the posterior fifth of this cord, near the processus vocalis, was a small sharply defined loss of substance.[1] A somewhat pinkish, broad-based, cauliflower-like mass protruded from the right ventricle. The entire aspect of the growths was very characteristic of multiple papillomata. The vocal cords could not meet on attempted phonation on account of the intervening masses, and the voice was reduced to a toneless whisper.

The large number of these small protuberances, their partly subglottic origin, and, above all, the well-known tendency of this class of growths to frequent and quick

[1] I have no explanation to offer of this ulceration. It looked as if artificially produced by attempts at operation, but no cutting instruments had ever—so far as could be ascertained—been introduced into the larynx.

recurrence, compelled me to form a very guarded opinion concerning the chances of even partial recovery of the voice.

The patient being nevertheless anxious for treatment, I, on the next day, in order to induce tolerance of instruments, used the laryngeal probe; and on the third day, I succeeded in removing, with the common laryngeal forceps, a large piece of the growth from the anterior commissure. Microscopic examination showed the neoplasm to be a villous structure, poorly supplied with blood-vessels, and covered with squamous epithelium. From this day forward I removed at nearly every sitting some portions of the supraglottic growths. At none of these operations was there any hæmorrhage.

But, as I had feared, recurrence soon became noticeable. Scarcely was a spot entirely freed from vegetations when, in spite of the application of strong nitrate of silver solution immediately after their removal, new protuberances made their appearance. The anterior part especially showed extraordinary powers of regeneration, and I had to remove portions from the anterior commissure four times in three weeks. But the power of reproduction became more sluggish each time, and was finally exhausted. Towards the end of July, the entire upper aperture of the larynx down to the level of the glottis was cleared, and the patient could speak with a very gruff voice.

Now began the difficult part of the operation, owing to the remaining growths being placed *underneath* the right vocal cord, and protruding but little beyond its border into the glottic space. The forceps, instead of seizing the growths, pressed away the vocal cord *and* the growths beneath it. It soon became evident that this method was not likely to be successful, and it therefore was necessary to adopt some other procedure. The knife, guillotine, écraseur, and galvano-caustic loop, were apparently equally ill calculated to remove growths springing from the under surface of a vocal cord.

In these circumstances I devised a specially adapted instrument. I bent the pointed endolaryngeal galvanocautery of Schech rectangularly, in such a way that the angle was situated in the *copper* part of the instrument; in other words, so that the projecting sheath projected below the elbow. This contrivance was then, by the help of the mirror, passed into the glottis during a deep inspiration, the horizontal part being held in the median line of the body. Having passed it below the glottis, I gave it a quarter turn to the right, and, raising the instrument slightly, brought its horizontal branch (only the tip of which consisted of platinum) against the roots of the growths which densely covered the under surface of the right vocal cord. I then burned the mass superficially. The spasmodic closure of the glottis, which invariably occurred as soon as the horizontal branch of the instrument came in contact with the under surface of the left vocal cord, and the approximation of the two cords during the cauterization, did not in the least interfere with the procedure, because the inner border of the *left* vocal cord never came in contact with the glowing platinum, but only with the warm copper portion of the instrument. No œdema or other troublesome symptoms followed either this first or the subsequent applications of the glowing wire. The operation was frequently repeated during the next weeks. The growths gradually disappeared, there seemed to be no tendency to recurrence of the subglottic portion, the patient's voice recovered more and more its tone and clearness, and in the beginning of November, 1881, she was practically cured, a scarcely noticeable want of clearness in the voice being no doubt due to the little gap on the inner border of the left vocal cord already referred to, and to some congestion of that cord. Since then no sign of reproduction has been manifested, and the patient's voice has become quite clear. The young lady was seen at the beginning and end of the treatment by Drs. George Johnson and F. de Havilland Hall.

CASE 2. *Large, hard, vascular fibroma, occupying the entire glottic space, springing from the anterior commissure of the vocal cords and the anterior third of the right vocal cord, and attached by a broad base; complete aphonia, strong dyspnœa; removal by galvano-caustic loop.*— Martin Sch., æt. 38, a German master-baker, came to me on September 29th, 1881, with complete loss of voice and dyspnœa, aggravated on exertion. Ten years ago, he began to be hoarse with a little cough. This had gradually increased to the present time. About four years ago, his breath became short when he walked about or went upstairs. For three years, he had felt, on deep inspiration and on coughing, something move slightly up and down in his throat. He was sure that he had never coughed anything out. The diagnosis of a growth in the larynx had been made two years ago by Mr. W. R. H. Stewart, who had also begun to practise treatment preparatory to its removal. But the patient soon neglected himself, and was not treated for his throat for the next two years.

On the 29th of September, 1881, the following was the state of the larynx, which, as in the first case, was the only organ affected.

A growth, as large as a small cherry, apparently commencing on the anterior commissure of the vocal cords, filled the glottic opening so completely that it was marvellous that the patient could still breathe.[1] The upper surface of the growth was irregularly globular, yellowish white, and richly supplied with blood-vessels, which showed as red streaks. The mass was very slightly moved during coughing and deep inspiration. Never did the smallest portion of the vocal cords become visible. Attempts to precisely define its attachment failed through

[1] It is a remarkable fact that obstruction to respiration, if *slowly* produced, may be carried to an extreme which, if *quickly* attained, is liable to cause dangerous or even fatal dyspnœa. Thus, in *chronic* bilateral paralysis of the abductors of the vocal cords, patients live still comparatively comfortably, even when the glottic opening is narrowed to a degree which has been practically shown to be quite insufficient for respiration, in cases of *acute* paralysis, produced within a few days.

the spasmodic coughing set up by the introduction of the probe. But as the hinder part was more mobile than the front part, it was evident that it was attached in the latter direction.

The appearances were so threatening that at first I thought of performing tracheotomy as soon as possible, as a prophylactic measure. But, on further consideration, I resolved to advise endolaryngeal operation in the first instance, while warning the patient that such a procedure, if not quickly successful, would be likely to hasten the necessity of tracheotomy. The patient placed himself entirely in my hands.

The passage for the breath being so very much narrowed, it was of paramount importance to enlarge this space as speedily as possible. But ordinary instrumentation would have been likely to cause swelling, which, even if slight, might have closed the air passages completely and imperilled life. Consequently I had to devise a plan by which, though intending to remove the growth *in toto*, I might have good prospect of removing at least a considerable part at the first attempt. Having to deal with a vascular, yet dense and probably largely adherent growth, the forceps and écraseur were unsuitable for my purpose, for either of them might set up free hæmorrhage, or tear away some of the healthy structures to which the growth was attached. The galvano-caustic loop was not open to these objections, while it might also be manipulated in a very small space. I therefore decided in favour of its use. Finally, I hoped to facilitate the operation by employing both of the two methods of local anæsthesia of the pharynx and larynx recommended by Schrötter and Rossbach. Thus Schrötter lessens the irritability of the pharynx and fauces by painting these parts repeatedly for several hours before operating on them, with a solution containing Potassii Bromid. ʒiij, Morph. Acet. gr. x, Glycerin. ʒij, Chloroform. ʒiij; whilst Rossbach directs an ether spray against a spot in the skin of the neck corresponding to the point of entry into

the larynx of the superior laryngeal nerve, with the view of causing anæsthesia of the mucous membrane of the larynx.

On November 11th, the first operation was performed. Drs. F. de Havilland Hall and George Mackern directed the ether spray simultaneously for nearly five minutes (Rossbach recommends only two minutes) against the spots corresponding to the entrances of the superior laryngeal nerves into the thyro-hyoid membrane, the patient's fauces and pharynx having been painted every half hour for two hours previously with Schrötter's solution. The lime-light illumination was used.

On the first occasion I removed a small piece from the hindermost edge of the growth; the patient thought that his breathing became at once easier, but inspection did not show that much space had been gained. The piece removed, when examined under the microscope, was seen to consist of bundles of fine wavy fibres, to be richly supplied with vessels, to be covered with squamous epithelium, and not to show any trace of cells. Subsequent examination of other portions of the growth confirmed this description. In spite of the use of the galvano-cautery there was comparatively speaking considerable hæmorrhage, and the patient coughed up on this and on the following day more light-coloured blood than had been observed in all the operations upon the case first related put together.

As to the local anæsthesia, I must say that, on this and subsequent occasions (at one of which Mr. R. W. Parker assisted me), it was not so marked as to compensate for the trouble it entailed; and I believe that the pharynx and larynx are rendered sufficiently insensible if the patient sucks small pieces of ice continuously for two hours before the operation. At the same time, I need not say that one observation would not justify a general conclusion as to the usefulness of a method, and I certainly intend to give Rossbach's plan another trial, if a suitable case should occur to me.

During the following weeks the operation was often re-

peated, but I succeeded only in removing small portions of the growth with the galvano-caustic loop. At last, however, I thought the airway sufficiently widened to allow me to attempt to tear away the growth with the forceps. But this method being followed by no better success and by more hæmorrhage, I allowed the patient a few weeks' rest, and then prepared a loop of much greater dimensions than before—so large, in fact, that I scarcely thought it would be possible to pass it into the larynx. But trying it on the 4th of January, 1882, I succeeded on the second attempt in ensnaring the whole mass, and after heating the loop with the galvanic current, I felt the growth come away with the loop. The tumour being left in the patient's mouth was expelled by coughing. The cauterisation was *absolutely painless*, for the patient, who always felt when I had got hold of the growth with either loop or forceps, was greatly astonished at seeing the tumour come away.

The growth is the largest I have ever seen, and, judging from recorded cases, probably one of the largest benign laryngeal tumours ever observed. When recent it measured more than half an inch by its long diameter and an inch and a quarter in circumference. It had been cut through very cleanly, and only a *red* heat having been used on this occasion, there was scarcely any hæmorrhage. The patient was greatly relieved, and his voice, though still very hoarse, increased in strength at once. A remaining portion of the growth, the size of a flattened pea, was removed with cutting forceps on February 10th, and the glottis was thenceforth perfectly free, although a very slight thickening persisted at the anterior third of the right vocal cord, indicating the point of attachment of the growth.

As this variety of tumour does not recur, and as the voice is now quite normal, the patient may be said to be cured.

Remarks.—In the preface to Paul Bruns' excellent

work, 'Laryngotomy (meaning Crico-Thyrotomy) for the Removal of Laryngeal Growths,' it is said that a warm controversy is now being maintained between surgeons and laryngologists as to the propriety of dealing with laryngeal tumours from within the mouth or from without, a controversy in which as yet no reconciliation of conflicting opinions has been arrived at. This observation, though written in 1878, with slight modifications still holds good, and particularly in this country.

It is true that for some years past the advocacy of the exclusive treatment of benign tumours by external operations has been less urgent, and surgeons now admit that the plan of removal through the mouth may be applied to a greater variety of cases than was allowed in 1872, when the late Professor Hüter, of Greifswald, taught that only small pedunculated solitary neoplasms springing from the free border of a vocal cord were suitable objects for endolaryngeal interference. This admission, however, is still only partial and tacit.

The conditions which in the opinion of many surgeons serve as contra-indications to endolaryngeal operations are—*broad bases, vascularity, and hard consistence of the neoplasm, its subglottic position, large size, multiplicity, and tendency towards recurrence.* With regard to all these points, I hope that the two cases just narrated will be regarded as *practical* proofs against the validity of any objection to endolaryngeal interference founded on them.

As regards my second case, in which I was very doubtful whether it would be possible to proceed endolaryngeally, it is not the first of the kind reported, and in several of these cases quite as successful results were obtained.

It may be asked, "Why was not thyrotomy performed in this case, as it would have led to a complete removal within a shorter time?" The following were my reasons:

First. The abbreviation of the time of treatment by thyrotomy is an illusory advantage. My patient, it is true, by the plan of treatment adopted, had to attend between thirty and forty times until the growth was

extirpated, and the time of treatment extended over several months. But he was throughout that period able to follow his usual occupation and was not laid up for a single day. Thyrotomy on the other hand, supposing that not the slightest mishap had occurred and that convalescence had been uninterrupted, would have disabled him from attending to his business for at least a fortnight. No patient, I believe, before whom these two alternatives are laid, would hesitate in deciding for the former, even at the sacrifice of an hour out of each day's work.

Secondly. The advantages claimed for thyrotomy, viz. that it can be performed under all circumstances and by one operation, do not exist. In the first place, it has not always been possible to split the thyroid cartilage, and it is to be remembered that my patient had already passed the age at which ossification of the laryngeal cartilages takes place; and again, the supposed advantage of performing the operation at one sitting may become a great drawback, because it *must* be *finished* at one sitting, experience having shown that repetitions of the operation considerably lessen the chances of the recovery of the voice.

Thirdly. In many cases, sufficient access, even after *total* division of the larynx, is not obtained to permit of the complete extirpation of the growths. This important fact is proved by the comparatively large number of failures to effect their complete removal by thyrotomy.

But I lay the greatest stress upon a risk, which justifies great hesitation in performing thyrotomy in any case in which it is possible to avoid that operation. I refer to the great danger of *lasting impairment of the voice.* This danger, I believe, is much less appreciated than it deserves to be. That it is actual I shall at once proceed to prove; but I may be permitted to say, in passing, that if this is regarded by some as a matter of second-rate importance, patients are likely to hold a very different opinion. Patients suffering from grave forms of vocal impairment

often have considerable difficulty in obtaining situations and performing their duties; and it is easy to realise how serious a matter is the total or nearly total loss of voice.

Now, as to the disadvantages of thyrotomy in this respect, I cannot do better than quote Bruns' conclusions; which are incontestable, because, on the one hand, his material includes *all* the cases on record when he wrote, and because, on the other, he carefully excludes from his statistics all those cases of alteration or loss of voice in which such changes could *possibly* be attributed to any other cause than the operation itself. Thus, out of the 97 cases of thyrotomy for the removal of benign growths on record up to 1878, only 38 could be used for the decision of the question whether thyrotomy is *in itself* dangerous to the vocal function. Of this danger there can be no doubt, for in only 18 out of the 38 cases was a normal or nearly normal voice restored or retained, while in 20 cases, that is, in the larger half, the voice was either completely lost (6 cases) or reduced to nearly complete aphonia or extreme hoarseness (14 cases). If, as Bruns remarks, *all* the cases then on record are compared as to the restoration or non-restoration of the voice, without making allowance for special circumstances, there appear to be *more than three times* as many failures as successes.

One word as to my non-performance of prophylactic tracheotomy in this second case. The result is in itself a proof that it was not imperatively demanded, but I certainly should not condemn the practice in such a case as this. The better course is to explain to a patient that if he declined to have an endolaryngeal operation performed, tracheotomy would under any circumstances soon become unavoidable; that by timely submitting to endolaryngeal interference, the external opening of the air passages might possibly be entirely avoided; but that, on the other hand, endolaryngeal interference, if not soon successful, might cause a certain amount of congestion, contribute to augment the already existing stenosis, and

thus possibly hasten the necessity of performing tracheotomy.

As regards my first case, its main interest lies in the multiplicity, the partly subglottic position, and the tendency to recurrence of the papillomata. The case practically shows that the *combined* occurrence of all these three circumstances, by some regarded as so many contra-indications to endolaryngeal removal, does not constitute an insurmountable impediment to the adoption of that method, any more than did in the first case the *combination* of large size, broad attachment, great density, and vascularity of the growth. Removal from within is, moreover, certainly preferable to thyrotomy in cases of papilloma, so far as relates to the probability of recurrence. For Bruns' tables show that after thyrotomy performed for multiple papillomata in adults and children, out of 39 cases, there were 18 cures and 21 recurrences, whereas after endolaryngeal operations under the same conditions there were out of 90 cases, 60 cures and 30 recurrences. In other words, *whilst thyrotomy was followed by a few more recurrences than cures, the endolaryngeal method obtained twice as many cures as recurrences.* These numbers are opposed to any belief in the protection against recurrence supposed to result from thyrotomy.

It is certainly remarkable that the tendency to recurrence should have so evidently become exhausted in my first case. Taken by itself, it affords a refutation to the wholesale condemnation of endolaryngeal interference in similar cases, a condemnation partly based also upon a fear lest innocent papilloma should, by protracted local irritation, be converted into epitheliomata. But I refrain from entering on this question on the present occasion.

It remains to consider the employment of the galvanocaustic method in both of my cases. The question as to the use of this method in the upper air-passages was discussed in the Subsection for Diseases of the Throat of the late International Congress (vide 'Transactions,' vol. iii, pp. 267—277). There was an almost unanimous

agreement as to its utility in certain diseases of the pharynx and nose, but less so as to its use in the larynx.

My own opinion with regard to this question is this. As a matter of principle I would never use the galvano-cautery in the larynx as long as simpler methods would suffice. Considering further how much depends upon the co-operation of the patient himself in all endo-laryngeal operations, I should certainly not make use of the galvano-cautery—however desirable for other reasons its employment might be—if I had to operate on a very nervous patient. Finally, I would urge the *great* importance of using a perfect apparatus. Too much has been said of possible dangers, such as the production of œdema and the burning of adjacent healthy parts. It is evident that the coincidence of all those circumstances, which make the use of the galvano-caustic method in the larynx not only desirable but also expedient, is likely to remain comparatively rare. On the other hand, cases are sure to occur occasionally in which the galvano-caustic method cannot be replaced with equal advantage by any other procedure.

In conclusion, I wish to repeat, that in bringing forward these two cases my object has been to show that even those which are believed by many laryngologists to be unsuitable for endolaryngeal interference—*i. e.* cases in which a *combination* exists of several of the above-enumerated difficulties—may with advantage be treated in that way. It is of course impossible to lay down absolute rules which should govern one's decisions as to the choice of method in every case of laryngeal growth. Each case, as a rule, shows so many features individual to itself that it should be judged on its own merits. *Exclusive* adherence to *endo*laryngeal operations would scarcely be less open to objection than the too conservative clinging to the maxim that benign laryngeal growths belonged to the sphere of *extra*laryngeal surgery.

DESCRIPTION OF PLATE VII.

Two Cases of Laryngeal Growths, in which these growths were successfully removed by Endolaryngeal, mainly Galvano-caustic, Operations. (FELIX SEMON, M.D.)

CASE I.—Multiple, sessile, in part subglottic, recurrent papillomata.

Fig. 1.—The larynx when first seen.

Fig. 2.—The same larynx after removal of the supraglottic portion of the growth, showing the papillomata underneath the right vocal cord.

Fig. 3.—The same larynx at present, after galvano-caustic removal of the subglottic papillomata.

CASE II.—A large, hard, broad-based fibroma, originating from the anterior commissure of the vocal cords, and from the right vocal cord (anterior third).

Fig. 4.—The larynx when first seen.

Fig. 5.—The body of the growth immediately after removal (natural size). The lower (red) surface shows the clean cut effected by the galvano-caustic loop.

Fig. 6.—The same larynx at present. The slight thickening of the anterior part of the right vocal cord marks the spot from which the growth originated.

Case . 1 .

Fig. 1.

Fig. 2.

Fig. 3.

Case . 2.

Fig. 4.

Fig. 5.

Fig. 6.

ON THYROTOMY

FOR THE

REMOVAL OF FOREIGN BODIES IMPACTED IN THE INTERIOR OF THE THYROID CARTILAGE.

BY

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All operations for the removal of foreign bodies from the air-passages are matters of interest to the practical surgeon, and any experience which has been obtained of comparatively new proceedings is valuable as guiding our course in these grave and sometimes very difficult emergencies. Now, the operation of thyrotomy, as it is called, or, as it may possibly be better termed, "total laryngotomy" or "chondro-laryngotomy," is a comparatively modern proceeding. All these operations through the thyroid cartilage,[1] as far as I know, have been performed within living memory. Gibb, in 1865, spoke of his case[2] as being the first operation of thyrotomy ever performed in England. Since then no doubt the operation has

[1] A case is erroneously ascribed by Planchon to Pelletan, but the original clearly states that the incision was made through the cricoid cartilage, not the thyroid.

[2] 'Brit. Med. Journ.,' September 30th, 1865.

become common, but it is practised usually for the removal of tumours; and both in this class of cases and in those of foreign bodies lodged above the glottis its application is now much restricted by the use of the laryngeal forceps and snare.

I propose in this paper to discuss the operation only so far as it is applicable to the removal of foreign bodies. I will first give short notes of a case which lately occurred in my own practice, and which was interesting as enabling us to see the condition of the parts subsequent to the operation after nearly complete union had taken place, and I will then discuss the indications for the operation, the best method of performing it, and its results so far as I have been able to discover them.

Case.—Bernard M—, æt. 30, a large, muscular man, was admitted on November 3rd, 1881, with a foreign body impacted in the larynx. About two hours before, he was eating some rabbit soup, when some one made him laugh and a piece of bone which was in the soup got into his windpipe. He had violent dyspnœa for a few seconds, but coughing seemed to displace the foreign body, and he was somewhat relieved. When admitted, he was breathing quite freely, the voice was a husky whisper, and he pointed to the left side of the thyroid cartilage as the seat of his trouble. Laryngoscopic examination was quite easy, and showed a piece of bone lying parallel to the left vocal cord. The bone was judged to be in the ventricle of the larynx. An attempt was made by the house surgeon to remove it with laryngeal forceps, and these attempts were repeated during the next few days by Dr. Whipham with forceps and bent probes of various forms; but though the bone could be touched, and though a small piece was on one occasion broken off, it could not be moved. Meanwhile the man was getting more distressed. There was a copious secretion of frothy sputa even from the day after the accident, and this became more abundant. The mucous membrane above the cord became much swollen, especially on the left side, and patches of white

fur began to appear on the base of the tongue and on the laryngeal mucous membrane.

As it was clear that the foreign substance could not be successfully dealt with without tracheotomy, I performed that operation on November 8th, making a free incision through the cricoid and upper rings of the trachea. As considerable hæmorrhage took place from a large vein, which ceased on the insertion of the laryngeal tampon, it was decided to leave the case for a time, hoping that the foreign substance might now become less firmly impacted or might be pushed up from the wound. Dr. Whipham examined the patient next day, and reported that the bone was in the same place, and so firmly impacted and buried in the swollen mucous membrane that its removal from the mouth was impossible.

On November 10th (a week after the accident), I performed thyrotomy. After making an unsuccessful attempt to seize or to displace the bone from the tracheal wound, the parts were carefully dissected off the thyroid cartilage, and the middle line of the pomum Adami carefully followed from the top to the bottom, till the knife touched the tracheal canula. Then the lips of the wound were held open, and the finger at once came down on the bone, which was extracted without the least difficulty. It is seen to be of large size (about ¾ of an inch square), bent at right angles, and with a very sharp spine projecting from one corner. It looks like part of the base of the rabbit's skull. The operation was greatly facilitated by the use Dr. Semon's modification of Trendlenburg's tampon, which prevented the entrance of any blood into the trachea. The bleeding, however, was not at all formidable. After renewed search with the finger had proved that there were no fragments left behind, the soft parts were brought together with silver sutures. Two hours after the operation the air was let out of the tampon, and on the follow-morning the tracheal tube was altogether removed in order to free the windpipe from all irritation of foreign bodies. The man slept well, his breathing was unem-

barrassed, and he was free from pain. Still there was very profuse expectoration, obviously from tracheitis, for there were no bronchial sounds. It was not till a week after the operation that the vocal cords could be seen from the mouth, and then only indistinctly, on account of the swollen membrane above them. The patient, however, seemed going on well. All the portion of the wound above the passage of the tracheal canula healed very rapidly, and the lower part of it slowly contracted.

On November 23rd (thirteen days after operation) he had a rigor, and the temperature, which had been normal, rose to 103° F., but fell again to the normal next day. It seemed that he had had ague previously, and it was doubted whether this was some irregular aguish symptom. It was also discovered that some food had been smuggled in by his friends. At any rate, there was not at first any cause for serious alarm, but the persistence of the frothy expectoration was disappointing. The throat was sprayed with carbolic acid and then with alum, but with little result. The voice, meanwhile, was improving, and the wound had contracted to a very small chink, through which, however, the air still passed. No morbid sounds were detected in the lungs, forty-eight days after the operation.

Things went on in this way till December 28th, when he had a severe rigor while sitting before the fire, followed by a second in the middle of the night; and now evidences of consolidation were found in the right lung, and of congestion in the left, and he complained of pain in the right side of the chest. The temperature would rise usually to about 100° at night, and the cough was very troublesome. On January 11th he brought up a considerable amount of blood, and now the sputa were observed to be extremely offensive. The hæmoptysis recurred on the evening of the 14th, followed by the expectoration of a quantity of foul pus, and soon after this he quietly expired, rather more than nine weeks after the operation.

The larynx was removed from the body, and is now

exhibited to the Society. The only thing noticeable in it is that the left vocal cord is slightly ulcerated. The line of incision on the inside of the larynx is so perfectly united as to be hardly perceptible, except at a point about the centre of the thyroid cartilage, where there is a minute opening. The external wound was also quite closed, except one small granulation, the position of which did not correspond to the unhealed part of the thyroid cartilage. The vocal cords are on the same level, in fact, it is hardly possible to see that they have been ever disturbed. The incision reached from the upper border of the thyroid to the first or second ring of the trachea, but the lower part of it was so perfectly healed that it is hardly possible to be sure whether these smaller cartilages had been divided.

The trachea and bronchi were much congested. The base of the right lung was occupied by a large foul-smelling gangrenous abscess. The left lung was greatly congested.

The liver weighed six pounds, and was cirrhosed to a great extent. The spleen was soft and large, the kidneys much congested, but not showing any evidence of structural change. The other organs were healthy.

The issue of this case was very disappointing, and it is still to me difficult of explanation. No doubt the man was unhealthy, and his liver bore evidence to his having been of intemperate habits. Yet this seems of itself rather an inadequate reason for death ensuing on so comparatively trivial a lesion as the lodgment during seven days of a foreign substance outside the true larynx. It will be noticed that except at the first moment of the accident, when the bone no doubt passed down to the glottis, and probably one of its pointed edges excited the cords to spasm, he had never suffered from any urgent dyspnœa, and therefore it seemed wrong to be in a hurry to open the windpipe so long as there seemed the least chance of extracting the foreign substance through the mouth; and equally wrong, after the tracheotomy, to be

in a hurry to divide the thyroid cartilge until it had been rendered certain that the bone could neither be extracted nor dislodged through the tracheal wound. I may also observe that the view obtained through the laryngoscope did not lead us to think that the bone was either so large or so irregular as it proved to be. Only one edge of it was seen, and it was believed to be a mere spine stuck across the orifice parallel to and above the left true cord. In reality, however, there was a very large mass buried in the ventricle and in the swollen mucous membrane, and it seemed strange afterwards that the distress was not much greater. The comparative slightness of the symptoms and the assumed probability of removing the foreign body by milder means justified, I think, the delay which took place in performing thyrotomy. Yet, nevertheless, that delay was, I doubt not, the cause of death, by setting up a diffuse or spreading inflammation of the respiratory mucous membrane, which the man's constitutional weakness rendered him unable to throw off.

It is surprising how rapidly in cases of lodgment of bones, or similar pointed substances, the inflammation will spread along the mucous surface. A child was brought to St. George's Hospital under my care some years since, whose case exactly resembled the present one, so far as the accident went, though the result was more fortunate. He had got a piece of rabbit bone into the larynx while eating some soup. When admitted, only a few hours after the accident, there were loud fluid râles over both sides, and when the bone was extracted (which was done with forceps from the mouth under chloroform) in the evening, the râles had spread quite to base of both lungs. In that case a large prong was found sticking out of the chief mass of the bone, and probably projecting through the glottis and irritating the mucous membrane. In all such cases the removal of the foreign substance is urgent. But in my patient we could not hear any morbid sounds in the thorax, and therefore thought ourselves justified in waiting. And no doubt,

when the foreign body can be seen and touched, and when there are no serious spasms, it can usually be extracted from the mouth. Examples of this kind are found in the works of Mackenzie. As a good example of success in a case very similar to the above I would refer to that recorded by Dr. Morell Mackenzie,[1] where a piece of bone was impacted transversely in the larynx above the vocal cords, and where, after fruitless attempts to remove it, tracheotomy was performed three days after the accident. Various fresh attempts were then made, and thyrotomy was contemplated, but at length, on the fifteenth day after the accident, the bone was broken with the laryngeal probe, and soon afterwards the rest of it was coughed up, and the man at once recovered.

This is by no means the longest period that a piece of bone has remained innocuously in the larynx. Planchon[2] quotes a case from the 'Gaz. Med.,' 1868, in which tracheotomy was practised seven days after the accident. It was not till the nineteenth day that the surgeon was able to assure himself of the position of the foreign body, and it was not till seven weeks after this that he decided to open the larynx, "fearing," as the report says, "that the vocal cords might become ulcerated." Notwithstanding that the bone had thus remained in close proximity to the vocal cords for nearly ten weeks, the patient recovered entirely and regained his voice.

Still more remarkable is the following:

In the 'New York Med. Rev.,' Aug. 15th, 1870, Dr. Gurdon Buck relates the case of a lady, æt. 25, in whom a fishbone had been lodged somewhere near the ventricle of the larynx for about two years and a half, causing great distress and constant cough. The diagnosis was long doubtful, but at length it was decided to lay open the larynx. This was done by first incising the crico-thyroid membrane, in the middle line, then continuing the incision through the lower half of the thyroid cartilage,

[1] 'Diseases of the Throat and Nose,' i, p. 417.

[2] 'Faits cliniques de Laryngotomie,' p. 68.

and as soon as respiration was established freely through this wound, completing the division of the thyroid cartilage. As no foreign body could be found the wound was extended through the two upper rings of the trachea and a tube introduced, for which purpose it was thought necessary to cut away part of the tracheal cartilages. It is not necessary to follow this interesting case in all its details. Suffice it to say that repeated examinations failed to detect the bone, though it was believed that there was an ulcerated patch of membrane on the interior of the cricoid cartilage. The patient had to wear a tube permanently, and it was not till twelve years afterwards, *i.e.* more than fourteen years after the accident, that the fishbone presented itself in the fenestra of the tracheotomy tube, and it was not till a year after this that she would consent to the removal of the tube, after which a plastic operation was necessary to close the wound. She recovered completely, but unfortunately no exact details are given of the final condition of the voice. Even before the removal of the tube, however, "all that was noticeable was a slight huskiness of the voice, and that by no means conspicuous."

In the face of cases such as these we can hardly say that the removal of a foreign body impacted above the vocal cords is a matter of immediate urgency unless it is exciting spasm of the glottis or giving rise to spreading bronchitis.

The next point is this: having made up his mind that operation is necessary, should the surgeon divide the thyroid cartilage at once, or should a preliminary laryngotomy or laryngo-tracheotomy be performed? Here we must distinguish between the cases in which the position of the body is known and those in which it is only suspected. In the latter there can be no doubt that an opening below the thyroid cartilage is the most prudent course. And for many reasons I think that even if the foreign substance is known to be lodged between the two alæ of the thyroid cartilage, it is better to make the

incision below it. However carefully performed, the division of the thyroid cartilage cannot but entail some risks to the voice. What those risks are will be discussed in the sequel, but I think every one will admit that there must be some danger to the integrity of the voice.

Now, it is clear from Mackenzie's and other cases where patience and perseverance succeeded in removing the foreign body without thyrotomy, that there is a fair prospect of the expulsion or removal of the foreign body by renewed attempts either from the mouth or the tracheal wound without any such dangerous interference with the framework of the glottis. Again, is it not possible that by making the incision directly on the foreign body through the thyroid cartilage the surgeon might displace it and drive it down below the wound, necessitating a further extensive incision into the windpipe under disadvantageous circumstances? The tampon in the trachea affords also an absolute security again hæmorrhage, and although the thyroid cartilage has been divided, without any preliminary opening below, and without any bad consequences, in several cases for the extraction of foreign bodies[1] as well as for the removal of tumours, yet I cannot but think the other course the safer, and the only argument against it is that a more extensive wound gives more freedom of motion for the two halves of the thyroid cartilage, and so renders the displacement of the vocal cords from each other more probable.

Another question of some interest relates to the possibility of extracting the foreign body by a partial division of the thyroid cartilage. This was successfully effected by Dr. E. H. Bennett, of Dublin,[2] in the case of a boy, æt. 8. Half a plumstone had lodged in or near the glottis. Tracheotomy was performed on the day after the accident, and many attempts were made to seize or dislodge the foreign body. This being found impossible,

[1] See Marjolin's, Maisonneuve's, Vidal's, and Blandin's cases as quoted by Planchon, lib. cit.

[2] 'Dublin Quarterly Journal,' August, 1870, p. 29.

partial thyrotomy was performed nineteen days after the accident, the lower part only of the cartilage being divided. This was completely successful, and in a fortnight the voice had entirely returned.

No doubt this partial division of the cartilage is less liable to be followed by displacement of the vocal cords, and in such a case as the above it may be well to follow Dr. Bennett's course. But where, as in my case, the foreign body is quite inaccessible from the crico-thyroid opening, and is known to be firmly impacted in the ventricle and buried in the swollen mucous membrane, it is hopeless to attempt its removal without dividing the entire cartilage, and thus attaining a free access to the parts above the glottis.

With regard to the results of the operation, I believe that the case which forms the text of this paper is the first in which death has followed the operation, unless we are to reckon as a fatal case one in which Marjolin operated[1] under a mistaken diagnosis. The foreign body (a bean) was really in the right bronchus, but the surgeon thinking that it was in the larynx laid open the thyroid cartilage. Death took place next day, but it does not appear from the report to have been due to the operation.

Mr. Durham, in a work on 'Injuries of the Air-passages," not yet published, says that "in at least eighteen cases it has proved successful, and that he does not find on record a single case in which death has resulted," reckoning Marjolin's case, no doubt, as one of death not from the operation, but from the unrelieved impaction of the foreign body.

I believe that this statement is essentially correct, though possibly the number may be open to question, since I find that Plauchon, who is one of the authorities on this subject, has admitted as instances of thyrotomy at least two cases in which the incision was made entirely below that cartilage. As far as I can discover, mine is the first case

[1] Planchon, Case 4.

in which death has followed after the operation of thyrotomy when performed for the removal of a foreign body, and even here it may very fairly be argued that it was not so much the operation as the delay of the operation which caused the man's death. He would doubtless have had a better chance of surviving had the operation been performed on the day of the accident.

The results as to the preservation of the voice seem in general to be very good. The dangers which threaten the integrity of the voice are two-fold, viz. from the ulceration of the vocal cords which the foreign body may produce, and from the irregularity of the cords which may follow on the operation.

As an instance of the former danger, and as a motive for speedy interference, I may refer to a case published by Dr. Stone in the 'Med. Times and Gaz.,' November 6th, 1880, and in which Mr. Croft operated. This case presents several points of interest. One very singular peculiarity was that though the foreign body (a piece of walnut shell which had been drawn into the larynx in a fit of epilepsy) could be easily touched with a probe from the laryngotomy wound, it could not be seen with the laryngoscope. In fact, laryngoscopic examination shortly after the accident showed the cords white and quite normal. Thyrotomy was not performed till twenty-six days after the occurrence, and then the piece of shell was easily extracted, but the boy has never been able to dispense with the tube, and (as Mr. Croft informs me) is still wearing one of india rubber. The strong probability in this case is that the cords were more or less ulcerated from the contact of the rough shell, which seems to have been buried in the ventricle.

Again, in the case related by Mackenzie,[1] in which Dr. S. Johnson extracted a "toy engine" which had been buried in the larynx for several months, the voice remained hoarse at the date of the report, three years after the operation. Such cases as these seem to show the

[1] 'Dis. of Nose and Throat,' i, 412.

necessity for prompt interference in cases where the foreign body is known to be voluminous and rough, or where there is any good reason to think that it may be so. In my case it will be noticed that one of the vocal cords still showed distinct traces of ulceration, though the operation was performed only a week after the accident.

The dangers to the voice from the operation itself I cannot but think have been over-estimated. No doubt it is possible that the two halves of the thyroid cartilage may unite on different levels, so that the two cords would no longer meet quite evenly, and it is possible that the incision may deviate from the middle line, and that one of the cords may be wounded, and it is possible that the incision through the cartilage may not wholly unite, so that a fistulous opening may be left, or that a portion of the cartilage may die.

All these things are possible, but I see no evidence that any of them have ever taken place. I have not succeeded in finding any description of the appearances on dissection in any case where thyrotomy has been performed, and am inclined to believe that the preparation which I exhibit to-day is in that respect unique. It shows really no deviation from the natural condition of the vocal cords depending on the operation, and without a somewhat close inspection there would be some difficulty in seeing that the larynx had ever been opened, so exact is the apposition of the parts, and so minute the perforation which remains in the thyroid cartilage. Clearly in this case the vocal cords are on the same level, and have not been interfered with by the incision, and though the wound has not entirely healed, there could be no doubt that it would soon have done so had the patient survived. Yet here the halves of the cartilage were not brought together with sutures, as is sometimes recommended (though, as I think, very erroneously), and the incision was to the greatest extent which is probably ever practised, viz. from the upper margin of the thyroid cartilage down to about the second ring of the trachea. The

patient had not entirely recovered his voice, but he could speak plainly and loudly, though somewhat huskily. This defect, however, depended, I have no doubt, on the irregularity of the left vocal cord caused by the impacted foreign body, and not on any result of the operation.

In numerous other cases the voice is reported as being perfect. Sometimes, as in Maisonneuve's, Vidal's, Coates's, Berr's,[1] the voice is only said in general terms to have been "restored" or "normal." In other cases, such as Mr. Durham's,[2] it is expressly stated that the patient "could laugh, talk, or sing, almost as well as ever;" and such, I think, we may fairly say will be the general result when thyrotomy is performed before the injury has produced ulceration of the vocal cords.

The conclusions, then, to which our present experience of thyrotomy for the removal of foreign bodies point, seem to me to be the following:

1. Very large substances may be impacted either in the ventricle of the larynx or between the two alæ of the thyroid cartilage, without causing any symptoms of immediate urgency.

2. When such foreign substances are rough or pointed they sometimes give rise to a spreading inflammation of the mucous membrane, and in such cases should be removed as soon as possible.

3. If they can be seen and touched they can usually be removed, either whole or piecemeal, with the laryngeal forceps.

4. When this is found impossible without tracheotomy, an opening should be made through the crico-thyroid membrane and upper rings of the trachea.

5. After this operation it is quite possible that the spasmodic condition of the parts about the glottis may subside, and a renewed attempt at extraction be successful.

6. If this is impossible the foreign body may be

[1] All these cases are to be found in Planchon's monograph.

[2] 'Guy's Hosp. Reports,' 1866.

either extracted or displaced from the tracheal wound, so that a preliminary tracheotomy is always advisable.

7. On the failure of such attempts the thyroid cartilage is to be laid open in the middle line partially, from below upwards, if the body is small and can be felt lying near the wound, entirely (and better from above downwards) if the body is large, firmly impacted, and lying altogether out of reach from the tracheotomy wound.

8. The operation of thyrotomy involves little danger to life, and not much to the integrity of the voice; at least, the risk of damage to the vocal cords is much greater from the protracted irritation of the foreign body than from the sequelæ of the operation.

CONCERNING THE ACTION

OF

SALTS OF POTASH, SODA, AND AMMONIA

ON THE

FROG'S HEART.

BY

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CAUSTIC POTASH, soda, and ammonia, also certain of the salts of potassium, sodium, and ammonium, were examined as to their action on the frog's heart, and found to affect this differently. A word of explanation as to the plan of the paper will be of service.

The mode of experimentation, and the results obtained with the hydrates and with the iodides, bromides, and chlorides of the above bases are first given. These results are then discussed, and the conclusions drawn. Finally, experiments with the citrates of potassium, sodium, and ammonium are given, and the results contrasted with those obtained with the previous salts.

The object in thus separating the salts was that the chlorides, bromides, and iodides might the better be considered apart from the rest as forming a group by themselves. In the table of quantities this plan has been departed from, the citrates being classed with the above salts; this is for the convenience of comparison and to avoid repetition.

The mode of experimentation was the following:—A Roy's tonometer was employed, the heart was tied on to the cannula as near as possible to, if not in, the auriculo-ventricular groove. (It is not always possible to avoid having a portion of the auricle below the ligature; this will be referred to later on when the experiments are discussed.)

As in previous experiments dried bullock's blood dissolved in water, so as to represent normal blood, was used; this was diluted in the earlier experiments with 2 parts of saline solution, $\frac{3}{4}$ per cent., in the later experiments with $2\frac{1}{2}$ parts. In the former 3 oz., in the latter about $3\frac{1}{2}$ oz. (more exactly 100 cubic centimètres), of this mixture were taken.

Where the action of the drugs was estimated quantitatively the dose was as far as possible kept uniform, and the times of addition also, the dose in question being added every quarter revolution of the cylinder (about 2—$2\frac{1}{2}$ minutes) to the whole mass of circulating fluid. The duration of each experiment was made as near an hour as possible, in order to reduce the error due to natural exhaustion which over lengthened periods would become considerable. The dose in consequence had to be adapted to this period. The tracings read from left to right.

The experiments were made in November, December, 1881, January and the early part of February, 1882.

The drugs were tested in two directions:

1. As to their action on the spontaneous working of the heart.

2. As to their action in modifying the effect of continuous faradisation applied to the heart.

The latter action will be first described, and this requires that the effect of continuous faradisation should be described.

Continued faradisation, provided the strength of the current be sufficient, and the interruptions frequent enough, causes in the normal heart a condition of continuous contraction, *i.e.* spasm, of greater or less extent.

To this persistent spasm—in which *fusion* of rhythmic contractions is the most obvious factor—the term "Tetanus" is here applied. As such it resembles closely the tetanus of a skeletal muscle, though differing in certain points.

Marey ('Physiologie experimentale,' vol. ii, p. 81, 1876[1]) we believe was one of the first to describe tetanus of the heart. He shows that the excitability of the heart is not the same at different moments of a cardiac cycle; thus a minimal stimulation administered during systole will not cause a contraction; the period during which this obtains is called the "refractory phase." The length of this period varies with the strength of the stimulus; the stronger the stimulus the less the duration, till the refractory phase quite disappears. Hence, when a series of *weak* electrical stimuli act on the heart, the greater number occurring during the refractory periods are ineffectual, and the number of systoles is much less than the number of excitations. But if the intensity of the current be increased (the frequency of interruption being unchanged), the refractory period lessening, the number of systoles approaches that of the excitations, and may equal them, a fusion results, *i.e.* a condition of tetanus.

Besides the influence of strength of the stimulus, Marey further shows that whilst cold increases heat diminishes the refractory period.

In our experiments, where continuous faradisation was

[1] Luciani ('Ludwig's Arbeiten,' 1872) had previously drawn attention to persistent spasm—the result of fusion of neighbouring beats—and had employed the word "*tetanus*." He shows that it may result as a first effect of the ligature, and also as a first effect of the circulation of serum.

employed, the cannula on which the heart was tied served as the one electrode, the other electrode pressed against the outer surface of the ventricle, care being taken that contact persisted throughout faradisation. The excitations were very frequent, the hammer of the induction apparatus vibrating rapidly.

The results obtained were in accordance with the previous statements from Marey, and it became clear that *fusion*, complete or incomplete, *i.e.* tetanus, complete or incomplete, could be produced in the following ways, viz.:

1. By lessening the refractory period. Increase of the strength of the current will effect this. Raising the temperature will do the same. Further, certain drugs will do the same.

Even in the cool months of October and November we met with hearts whose irritability was such that they exhibited this fusion from the onset. It was especially to this form of fusion that Luciani drew attention.

2. By prolonging the duration of each contraction. It is obvious that, other things remaining the same, increase in the duration of the beat must favour fusion. Some drugs, *e.g.* soda, ammonia, and its salts, probably act in part in this way.

3. Yet another way is to be noted. Certain drugs, *e.g.* soda, ammonia, potash, antiarin, digitalin, strophanthus, induce a persistent spasm of the ventricle, not, however, by means of a fusion of beats, for these may occur so infrequently as to exclude this mode; in such cases, even when the intervals between the beats are a minute or more, the persistent spasm may last during the whole period of the intervals. This persistent spasm will, *cæt. par.*, tend to fuse together neighbouring beats.

It has been suggested ('Journal of Physiology,' December,[1] 1881) that this spasm may be an increase of the tonus of the heart and allied to contracture.

[1] "Regarding the action of Hydrate of soda, ammonia and potash on the Frog's Ventricle."—Sydney Ringer, M.D.

The effect of temperature was tested, and it was found that whilst heat facilitated the production of tetanus, cold hindered such; these results are confirmatory of Marey's statements as to the refractory period. A difference of temperature amounting to 12° C. demonstrated the above very distinctly.

As to the effect of exhaustion, this does not appear to affect the facility with which the heart may be tetanised.

It is worth while contrasting cardiac tetanus with that of a skeletal muscle. In the latter case a stimulus sufficient to excite a good contraction will, if sufficiently frequently repeated, yield tetanus. In the former, a stimulus capable of causing full contraction of the heart, but not in excess of such strength, will not yield tetanus by repetition, however great the frequency attained. The explanation is to be found in the refractory phase attending each contraction. To get tetanus the strength of the current must be increased, whereby the refractory period is diminished up to extinction.

Diagram 1, fig. I, represents the development of tetanus. The figures above the line indicate the positions of the secondary coil of the Du Bois Reymond.

In A we have incomplete fusion; the frequency, though increased, is insufficient to effect complete fusion.

In B the effect of increased strength of current is seen to have caused greater frequency and completer fusion.

In C a further stage is shown, but here, with the secondary coil at 3, also with the coil at 5 in D, the individual beats can no longer be counted, the line being in parts practically straight.

In addition to increased frequency, both B and C show that the element, persistent spasm, is also present as a factor. The length of line above the trace represents the time of faradic excitation.

Having described the effect of continuous faradisation on the heart, we proceed to describe how this effect is modified under the influence of certain drugs.

The mode of experimentation was the following:—The

effect of faradisation was tried before the addition of the drug, and a certain degree of tetanus being selected to serve as standard, the addition of the drug was commenced; the secondary coil was maintained, of course, stationary, and at definite, equal intervals of time the heart was faradised. The intervals were eight or ten minutes in length, sufficient to allow the residual effects from the preceding tetanus to have passed off.

Hydrate of soda, hydrate of ammonia, hydrate of potash.

Hydrate of soda increases the readiness with which the heart answers to continuous faradisation. Thus, a strength of current capable only of slightly increasing the frequency of the normal beats, therefore short of producing tetanus, caused well-marked tetanus after the addition of soda. In this tetanus all three factors are probably at work, viz. diminished refractory period, persistent spasm (of the nature of tonus), increased duration of each beat. This increased readiness of response occurred without any diminution of the height of the trace. In the doses here used, then, soda increases what may be termed excitability, without affecting contractility (Diagram 1, fig. II, A, B, C).

From these observations, made in November, season (temperature ?) would seem to have a modifying influence. The dose of soda required to produce a given effect here was much larger than in summer, and the diastolic contraction, so well marked in summer, was much less developed, and then only after a relatively large dose.

Hydrate of ammonia.—The excitability to the continuous faradic is considerably increased, whilst the height of the beat is still undiminished. This increased excitability is maintained during the early part of the decline in height, then itself begins to decline, but even to the last the excitability remains, at least, as good as before the addition of ammonia (Fig. III, A, B, C, D).

The modifying action of season was marked here also. In summer a much smaller dose is requisite to develop

well-marked diastolic contraction and the persistent spasm indicated by departure from the base line, which spasm increasing, the ventricle finally stops in strong systole, in which condition it remains a considerable time. In the present series, made during November, the above effects were not strongly marked except a very large dose was administered.

Hydrate of potash.—The readiness to become tetanised is markedly decreased by potash, and a strength of current sufficient to give well-marked tetanus before addition subsequently soon loses its effect. A rather curious effect is further to be noted: not only may such current fail to show any tendency towards producing tetanus, it may actually suppress the spontaneous beats, these beginning again a little time after discontinuance of the faradisation (Diagram 2, fig. IV, A, B, C, D, E, illustrate potash). This suppression of the spontaneous beats is not shown in diagram.

Contrasting the effects of soda, ammonia, and potash, we have, on the one hand, soda and ammonia, which early and for a considerable period increase the excitability of the cardiac tissue, as tested by continuous faradisation; on the other hand, potash, the striking effect of which is a diminution of this excitability from the very commencement. This diminished excitability occurs, even though at the time there exist persistent spasm due to the drug (Fig. IV, C, shows this well).

It may be here stated in general terms that, so far as have been examined, the salts of potash act like the hydrate in the way in which they modify the effect of continuous faradisation, *i. e.* they lessen this from the commencement, and may further cause the suppression of spontaneous beats during faradisation above noted.

In the present series the iodide and citrate were tested on this point; the chloride had been previously examined (see the 'Practitioner' for January, 1882).[1]

[1] "Concerning the action of the Chlorides and Bromides of sodium, ammonium, and potassium on the Frog's Ventricle," by Sydney Ringer, M.D.

The fact of the citrate—an organic salt—conforming with the inorganic salts, the iodide and chloride, renders the presumption that salts of potash generally may behave alike in the above respect not improbable.

The other mode of testing the influence of drugs has now to be considered, viz. "*the action on the spontaneous working of the heart.*"

The drugs tested in this direction were of the bases—hydrate of potash alone—of the salts, the iodides, bromides, and chlorides of sodium, ammonium, and potassium, also the citrates of these three bases. Incidentally the modification of the effect of continuous faradisation was examined in the case of the iodides. The results of the experiments will be first detailed, and then the conclusions given to which these led.

The mode of experimentation has already been stated. The strength of the solutions employed was in each case 10 per cent. The quantities of this used are given in cubic centimètres, and the actual quantity of salt in grammes and grains. The quantity of blood mixture used was throughout 100 cubic centimètres.

The reason why of the bases, potassium hydrate alone was taken was that the tendency which the other bases—soda and ammonia—show to produce arrest of the heart in full systole, does not allow of a quantitative comparison between the drugs, since potash arrests in diastole.

Caustic potash.—The salient feature here noted was the persistent spasm excited, but, disregarding this, the effect on frequency, so far as the primary effect went, was inconstant, sometimes slowing, sometimes quickening, but in all cases marked slowing preceded complete inhibition, which occurred in all six cases, and on an average after 0·6 c.c. (= 0·9 grains) had been added and whilst the ordinate was of good value.

When in the final stage single stimuli caused no response, continuous faradisation was without effect.

With regard to the height of the beat, this was not much affected in the early stage, but chiefly towards the

end, and in particular after complete inhibition. No primary increase in height was noted such as ammonia shows. The persistent spasm or diastolic contraction, which has been previously described ('Journal of Physiology,' Dec., 1881), was here particularly well marked.

The quantities required to arrest the heart were:

Exp. I.—Feb. 7.	Temp. of room	15° C.	Quantity	0·75 c.c.
„ II.— „ 7.	„	15·5°.	„	0·85
„ III.— „ 8.	„	15°.	„	1·05
„ IV.— „ 9.	„	14·5°.	„	1·05
„ V.— „ 9.	„	15°.	„	0·95
„ VI.— „ 10.	„	15°.	„	0·65
				5·3

∴ Average 0·88 c.c. = 0·088 grms. = 1·35 grains.

Iodide of sodium.—The effect on the rhythm observed here was a slight lessening of the frequency of the beats, but in no case were these arrested, so that the beats remained spontaneous to the very end, *i.e.* as long as contractility persisted. The heart is arrested in diastole.

In the final stage, when the beats had quite disappeared or showed only as a faint waviness of the trace, and single induction shocks would either give no response or one only just visible, continued faradisation of the same strength or weaker would cause persistent spasm, reaching a maximum by successive steps, each contraction being piled up, as it were, on the top of the preceding one. (See Diagram 3, fig. VII A.

In connection with this it must be mentioned that occasionally, whilst the heart was beating spontaneously but under the influence of the sodium salt, a somewhat similar piling up was observed, occurring without obvious cause.

This phenomenon has been referred to throughout this paper, and the expression "*piling up*" retained, but it must be stated that this mounting up by a succession of steps is not always seen; the term has, however, been kept, and for this reason it must be remembered that the essential part of the phenomenon is the height attained,

which is far in excess of any individual contraction spontaneous or excited. (See fig. VII, A and B.)

As to the effect on contractility the amplitude of the beat gradually lessens, but in the end stages, when the height has been greatly reduced, the breadth of the trace, *i.e.* the duration of the beat, becomes increased, in some cases very considerably.

Iodide of sodium is very little poisonous, as will be seen from the quantities appended, which were those requisite to stop the heart:

Exp. I.—42 c.c.	Temp. at time of experiment		12·5° C.
„ II.—72	„	„	10·5°
„ III.—48	„	„	10°
„ IV.—54	„	„	9·5°
„ V.—52	„	„	9°
„ VI.—60	„	„	8·5°
328			

Average 54·6 c.c. = 5·46 grms. = 84·24 grains.

Iodide of sodium lessens in a marked degree the excitability to continuous faradisation.

Iodide of ammonia.—This is an unstable salt, readily yielding free ammonia and iodine. A good commercial specimen was used, containing, however, traces of the above.

The effect on rhythm is not marked, spontaneous beats continuing till contractility is nearly, if not quite, destroyed; in this respect ammonium iodide agrees with the corresponding sodium salt. The heart is arrested in diastole.

After the heart has been arrested, neither single induction shocks, nor continuous faradisation, has any effect. In this respect the salt contrasts with sodium iodide.

The heart is arrested by the destruction of contractility, the beats growing weaker and weaker till they finally disappear. There is, however, probably in all cases an early stage during which the ventricle empties itself more completely, *i. e.* a stage of stimulation; this is scarcely appreciable when the ventricle is contracting vigorously before

addition of the drug, but when failing to empty itself quite, the above stage is indicated by increased amplitude of the trace, and with this increased height rounding of the top of the trace is noted. If a large dose be suddenly added, both diastolic contraction and persistent spasm appear. In one case, as the effect of a large dose, spontaneous beats were arrested and replaced by a slowly remitting spasm.

This salt is a far more powerful paralyser of the ventricle than iodide of sodium. Six experiments gave the following numbers:

Exp. I.—4·6 c.c.	Temp. at time of experiment		12° C.
„ II.—4·9	„	„	12°
„ III.—4·2	„	„	13°
„ IV.—3·9	„	„	13·5°
„ V.—4·9	„	„	13·5°
„ VI.—2·1	„	„	14°
24·6			

Average 4·1 c.c. = 0·41 grms. = 6·3 grains.

Iodide of ammonium, like iodide of sodium, lessens in a marked degree the effect of the continuous faradic current. Suppression of the spontaneous beats during faradisation may even be witnessed (see Potash).

Iodide of potassium.—The striking feature in the action of this salt is that suddenly, whilst the height of the trace is still considerable, the spontaneous beats are arrested permanently, though there is still response to electric stimulation. If the addition of the drug be still continued the response, originally having the value of the last spontaneous beats, grows less and less till finally contractility disappears. Preceding the arrest of spontaneous beats these latter grow more infrequent. During lengthened intervals between spontaneous beats, or subsequently to permanent arrest, the less the interval since the last spontaneous or excited beat the stronger must be the excitation to be effectual. This of course between limits, for below a certain strength all excitations would be ineffectual, above a certain strength all would be effectual. The

same applies to a heart temporarily inhibited by the ligature. In fact, the ventricle arrested by iodide of potassium behaves in all respects like such heart.

When the contractility has disappeared to single induction shocks, continuous faradisation is without effect; in this respect it resembles iodide of ammonium, but contrasts with iodide of sodium.

Iodide of potassium further acts powerfully on contractility; this is seen, subsequent to arrest of the spontaneous beats, in the decreasing height of the excited contractions.

The following numbers were obtained:

Exp. I.—2 c.c.	Temp. 13° C.
„ II.—2	„ 15°
„ III.—2·2	„ 13°
„ IV.—2·2	„ 14·5°
„ V.—2	„ 15°
„ VI.—1·9	„ 9·5
12·3	

Average 2·05 c.c. = 0·205 grms. = 3·16 grains.

In its lessening the effect of continuous faradisation of the ventricle, iodide of potassium is similar to iodide of ammonium, but more powerful; as with this latter, in the final stages before complete arrest, the spontaneous beats become arrested during the faradisation, and a considerable interval may follow before the spontaneous beats recommence. In this latter respect it, with the ammonium salt, contrasts with sodium iodide.

A character shown in common by the iodides of all three bases was that, subsequently to the arrest of the ventricle by the drug, and when single electric shocks were without effect (also continuous faradisation in the case of ammonium and potassium iodides), the dilution of the circulating fluid with water (100 c.c. were in each case added) brought back spontaneous beats of good value; the recovery was but temporary, but in some cases a second and a third dilution (100 c.c.) were attended with

recovery, each time feebler and of shorter duration. With each recovery a certain amount of persistent spasm occurred, indicated by the trace retreating from the base line.

Bromide of sodium.—Six experiments were made. The effects were somewhat inconstant and anomalous; thus in two cases there was no inhibition whatever, the beats remaining spontaneous till contractility disappeared. These, then, were in accordance with the iodide and citrate of soda series. In one there was distinct inhibition, as marked as in the case of potassium salts. In the remaining three the rhythm was curiously affected, *e.g.* after the quantities had reached 15 c.c., 15 c.c., and 18 c.c. respectively, patches of beats separated by long intervals occurred, the end patch was of long duration, the beats frequent, and the rhythm persisting till contractility had disappeared. Though this phasic condition represents a form of inhibition, still it was not complete inhibition, and in five out of the six the final stages contrasted as markedly with the effects of potassium salts as did those of the other sodium salts.

In the final stage, when the strongest current excited either no response or only the faintest, the characteristic piling up was obtained with continuous faradisation. There was nothing special as to contractility. In the one instance of marked inhibition there was scarcely any reduction in height when this occurred.

The quantities were:

Jan. 19	.	.	.	Temp. 14·5°	...	42 c.c.
„ 24	.	.	.	„ 18°	...	32
„ 24	.	.	.	„ 17·5°	...	41
„ 26	.	.	.	„ 17°	...	34
„ 27	.	.	.	„ 17°	...	28
„ 27	.	.	.	„ 17°	...	24
						201

Average 33·5 = 3·35 grms. = 51·7 grains.

Bromide of ammonium.—The results obtained were very

uniform. In all, frequency was increased; in five, this occurred from the very beginning of addition of the drug, in one, rather marked slowing followed the first dose, but soon gave way to increased frequency. The increased frequency was maintained up to the end, disregarding some slight irregularity which in three cases appeared in the final stage.

Subsequent to the disappearance of spontaneous beats, there was no response to single shocks, and none to continuous faradisation.

On contractility, a primary effect in the way of increase of the height of the beat with broadening of the apex was distinct in some, very slight in others, but discoverable in all. Slight departure from the base line was also noticed, but not in all. The frequency of beats was too great to admit of diastolic contraction appearing. These last-named effects were early, viz. after the first or second dose. Fig. V A (Diagram 2) illustrates these effects.

The decline in height was rather rapid towards the end.

Quantities:

Jan. 28	.	.	Temp.	16·5°	...	Quantity	2·6 c.c.
„ 28	.	.	„	16°	...	„	2·4
„ 30	.	.	„	16°	...	„	2·2
„ 30	.	.	„	16·5°	...	„	2·4
„ 31	.	.	„	16°	...	„	2·6
„ 31	.	.	„	17°	...	„	2·2
							14·4

Average 2·4 c.c = 0·24 grms. = 3·70 grains.

Bromide of potassium.—In the six experiments made with this drug the effect in the way of inhibition was less marked than was the case with the other salts of potassium examined, viz. iodide and chloride. In general, moreover, there was less uniformity of action in the series.

In three out of the six complete inhibition obtained; in the remaining three, though in the final stages, the inhibition was almost complete, still, an occasional spontaneous beat occurred till the amplitude was practically abolished.

The effect, then, though somewhat less marked here, is still very decided.

In five out of the six cases slowing occurred as a primary effect, contractility suffering but slightly; the subsequent effects on rhythm were very irregular.

As to the effect of continuous faradisation, no piling up of the beats obtained as a final effect.

There is nothing special as to the effect on contractility excepting that the action hereon was not marked early. At the time of complete inhibition the beats were severally reduced to $\frac{3}{5}$, $\frac{1}{2}$, and rather less than $\frac{1}{2}$ their original height.

Quantities employed :

Jan. 19	. . 1·7	...	Temp. 15°
„ 20	. . 2·1	...	„ 17°
„ 20	. . 2·4	...	„ 18°
„ 21	. . 2·8	...	„ 14·5°
„ 21	. . 2·2	...	„ 15°
„ 23	. . 1·6	...	„ 13·5°
	12·8		

Average 2·13 c.c. = 0·213 grms. = 3·28 grains.

Of the bromide series the effect of dilution was tested with the ammonium and potassium salts. Dilution with blood mixture was substituted for dilution with water, 100 c.c. being added. Dilution was tried twice with ammonium bromide with negative result; no recovery occurred. With potassium bromide dilution in three cases brought back spontaneous beats of good height. The blood mixture was added when the ventricle had ceased to respond to the strongest stimulation.

Chlorides of sodium, ammonium, potassium.

Some experiments on these salts made in the summer months have already been recorded.[1] But as season

[1] 'Practitioner,' January, 1882.

(temperature ?) affects the action of remedies on the frog's heart the experiments were now repeated in January, that we might be able to compare the action of the chlorides with that of the iodides and bromides at the same time of the year.

Chloride of sodium.—As in the case of bromide of sodium, the effect on frequency was somewhat inconstant.

In one case, with the exception of trifling slowing, there was no effect on frequency, the beats remaining spontaneous to the end.

In two there occurred distinct inhibition quite of the potash type.

In a fourth marked slowing was produced, amounting in the end stages to inhibition, almost complete.

Lastly, in two cases the action of the ventricle became phasic, groups of beats alternating with intervals, this condition remaining to the end.

Here, though there was distinct disturbance of rhythm, spontaneous beats still persisted to the end. So that we have three cases of non-inhibition against three cases of inhibition.

Continuous faradisation, after arrest of the ventricle, gave the characteristic piling up.

The quantities required to arrest the heart were:

I.—Feb.	1.	Temp. of room	16°.	Quantity	16 c.c.
II.— „	1.	„	16·5°	„	14
III.— „	2.	„	—	„	20
IV.— „	4.	„	17°	„	23
V.— „	4.	„	17°	„	18
VI.— „	6.	„	9·5°	„	25
					116

Average 19·3 c.c. = 1·93 grms. = 29·77 grains.

Chloride of ammonium.—The effect on frequency was a slight increase of the contraction rate; the beats were accelerated on an average in the proportion of from three to five, three being the original frequency

In one case the beats became slightly slower throughout

but in this, as in all the others, spontaneous beats of good frequency persisted to the very last, *i.e.* till contractility disappeared.

When the heart had been arrested and single induction shocks caused no response, continuous faradisation was equally without effect.

This salt acts powerfully on contractility, the beats growing less and less till final cessation in diastole.

In all six experiments, as a first effect and for a short time the beats increased in height and became broader; in three this was very distinct, in the other three it was but slight; in the former there was also departure from the base line, showing a certain amount of persistent spasm.

The quantities required to arrest the heart were:

I.—Jan.	16.	Temp. of room	17° C.	Quantity	2·1 c.c.
II.— „	16.	„	18·5°	„	2·1
III.— „	17.	„	17°	„	2·
IV.— „	17.	„	16°	„	1·8
V.— „	18.	„	18°	„	1·8
VI.— „	18.	„	18°	„	1·8
					11·6

Average 1·9 c.c. = 0·19 grms. = 2·93 grains.

Chloride of potassium.—The first or second dose always caused slowing, then, in five out of the six cases, the beats again became more frequent, in some instances beyond the original frequency; then the beats again became slower with intervals, now amounting sometimes to from three to four minutes. This occurred on an average after 0·5 c.c. had been added. After as much as 0·76 c.c. = 1·17 grains, on an average, had been added, spontaneous beats ceased, though contractions could still be excited by induction shocks. This complete inhibition occurred when the beats were reduced respectively to $\frac{1}{3}$, $\frac{2}{5}$, $\frac{1}{5}$, $\frac{2}{5}$, $\frac{3}{4}$, $\frac{1}{6}$ of their original height.

When single shocks gave no result, continuous faradisation was equally without effect.

Contractility was completely destroyed after the addition of the following doses:

I.—Jan. 12.	Temp. of room	17° C.	Quantity	1·4 c.c.
II.— „ 12.	„	17°	„	1·5
III.— „ 13.	„	15·5°	„	1·8
IV.— „ 13.	„	16·5°	„	1·7
V.— „ 14.	„	19·5°	„	1·8
VI.— „ 14.	„	18·5°	„	1·6
				9·8

Average 1·6 c.c. = 0·16 grms. = 2·46 grains.

In respect of the discussion of the results obtained, it may be mentioned that this discussion has assumed the cardiac muscular tissue to possess the property of rhythmic contractility apart from ganglionic structures.

Since the paper was written Dr. Gaskell's paper, read before the Royal Society, has reopened this question. The results here obtained, however, as also the discussion of these, are in nowise affected by this question as to rhythmic contractility necessitating two structures, nervous and muscular, or but one structure, viz. muscular. That which is alone required here is a rhythmically contractile tissue; this given, we here show that the action of drugs on such demonstrates the dissociation of that which underlies "*contraction rate*" or "rhythm," from that which underlies "*contraction height.*"

The actual results obtained have been so far alone given; of these, certainly the most striking is this—that given a rhythmically contracting tissue, the action of a drug on this may show itself in two directions:

1st. As affecting the intervals separating successive beats.

2nd. As affecting the actual value of the beats themselves.

Thus we have seen drugs affecting the frequency or contraction rate, and also the height of the individual beats (this last has been always referred to as the action on contractility). The stress has fallen now in one direc-

tion, now in another, but in every case there has been some effect in both directions.

It was mentioned at the outset that the ligature was not always exactly in the auriculo-ventricular groove; this, however, does not introduce an error, for the action of the drugs was tested on a rhythmically contracting tissue, and this in all cases was secured.

The question now arises, are the two manifestations above mentioned intimately connected one with the other, so that action on the one necessarily involves action on the other? or may they be more or less dissociated from one another, and separately subject to influence? The former supposition, viz. that of an intimate connection underlying the dual manifestation, requires for proof evidence of such union in the shape of constancy of relation, both in kind and degree, *i.e.* qualitative and quantitative. If closely united, the evidence of such must be that they move together in *some* definite direction at *some* definite rate.

On the other hand, the proposition that they are dissociated requires the negative evidence of *absence* of any constant relation, qualitative or quantitative—the demonstration that they do not move together.

The consideration of this subject necessitates the use of *terms* in place of the phrases "value of each beat," "frequency of contraction." For the former the word *contractility*, as naming the underlying element, may be substituted; for the latter the term "*excitability*" has been selected. The term is not without objection, and must be taken in the wide significance of "*conditions antecedent to the contraction.*" These conditions are, of course, causal, but whether they be of the nature of "conditions generating the stimulus," or "conditions preparing the contractile tissue for the stimulus, *i.e.* rendering the stimulus available," must be left undiscussed. The terms being thus defined, we have to consider whether *excitability* and *contractility* must be associated or may be dissociated.

What do we actually find? In one case we note

increased frequency attending increased height of beat, in another increased frequency attending diminishing height; and examples of this are to be found not only in the case of different hearts, but even in the same heart it may be found. Thus the ammonia salts, *e.g.* the chloride or bromide, frequently show a primary increase of frequency even during the stage of increased height of beat, and this increased frequency is not only maintained but may even go on increasing as the height of the trace steadily falls. Thus, there is no constant qualitative relation between *contraction rate* and *contraction height.*

Again, in one and the same heart we note as the effect of a drug at one time marked effect, say on excitability, with no appreciable effect on contractility; a little later, and with no noticeable effect on excitability, the stress now falls on contractility.

Potash salts illustrate this very well. Thus, the first additions as a rule are followed by slowing, to the extent may be of doubling the length of the intervals, with scarcely any effect on the height of the beats; a little later on, and with almost unchanged rhythm, the beats may diminish by two thirds within the short space of seven beats.

Thus, then, even the same heart shows no quantitative relation that is constant between excitability and contractility.

One might multiply evidence in the same direction. Thus, quite suddenly a heart, beating at intervals of fifteen to twenty seconds, will start off at a rate five to ten times as fast, with little if any variation in height of beat. But sufficient has been said to prove that, whatever underlies these two manifestations, which are measured by *contraction rate* and *contraction height,* they may be separately influenced, and therefore so far are distinct.

Hence one may speak of a drug as acting on either *excitability* or *contractility,* and the results already given may be more briefly and clearly summed up as follows:

In the case of the drugs already examined, and probably in all cases, the action is not exclusively on either *excitability* or on *contractility; both* are affected.

The degree, however, in which one or other suffers varies with different drugs. Thus, with regard to the salts of the three different bases, sodium, ammonium, potassium, the effect on excitability varies greatly; and, whilst potassium salts strongly affect excitability, sodium and ammonium salts affect excitability relatively but slightly. And thus, whilst with potassium salts it was the exception not to get permanent arrest of spontaneous beats before contractility was destroyed, with sodium and ammonium salts it was the exception when spontaneous beats did not continue up to the very end, and, moreover, with a final frequency little short, often in excess, of the original frequency.

Though there is this broad division into potassium salts on the one hand and ammonium and sodium salts on the other, the salts of ammonium and potassium form the extremes, those of sodium being intermediate, affecting as they do excitability rather more than ammonium. As to the action on contractility, the quantities of the drugs used constitute the measure of this action, since in each case the contractility was reduced to *nil*. On examining the table of quantities it will be seen that a very different relation now obtains. Potassium and ammonium come very close together, whilst sodium is widely separated. The two former are so near numerically that it would be unsafe to draw inferences from the differences in the actual numbers; in passing to sodium the highest estimate, in the case of the chloride, bromide, and iodide, would give the relation as one to ten, the sodium salts being one tenth as poisonous as those of ammonium and potassium.

Table of quantities.

Potassium hydrate . . .						Quantity in grs. . 1·35	
	Chloride. Quantity in grs.		Bromide. Quantity in grs.		Iodide. Quantity in grs.		Relation of molecular weights.
Sodium .	29·77	...	51·7	...	84·24	...	58·5 : 103 : 150
Ammonium	2·93	...	3·7	...	6·3	...	53·5 : 98 : 145
Potassium	2·46	...	3·28	...	3·16	...	74·5 : 119 : 166

Citrate.			Molecular weights having equal number of atoms of base.
Sodium	. .	16·66	... 357
Ammonium	.	3·3	... 339
Potassium	.	2·46	... 324

In the above the only column which perhaps requires explanation is that giving the relation of the molecular weights. The object of this was to see, if, taking a given base, any relation could be traced between the action of its salts as here tested and the molecular weights of the same salts.

There does appear to be some such relation in the case of the sodium salts of chlorine, bromine, and iodine. But this fails in the case of the ammonium and potassium salts, and so, the results obtained do not point in any one direction. Obviously, however, a very much larger number of experiments would be needed to arrive at any definite conclusions, positive or negative, on this point.

One must be careful not to take effect on contractilty as the exclusive measure of poisonous action.

Arrest of the contractions rather should be taken to represent the poisonous action, and this we have seen may happen in two ways, by the action on *contractility*, and by the action on *excitability*. Hence though as to the first ammonium and potassium show but little difference, as to the latter they diverge widely, and potassium stands as by far the more powerful poison.

The recognition of the above has especial importance from a clinical standpoint, for, if we be dealing with the functions of an organ, the arrest of such functions concern us more immediately than the precise mode in which such arrest is effected. And if a drug threaten in two directions, the dosage must take both of these into account.

Accordingly, represented in descending order, we have:

Potassium salts, most poisonous, both *excitability* and *contractility* powerfully affected.

Ammonium salts, next in order, *contractility* suffers almost alone.

Sodium salts, least poisonous, *contractility* suffers chiefly, but *excitability* is more affected than in the case of ammonium salts.

Further, it must be remembered that sodium salts are not only least poisonous of the three, but are, indeed, very weak poisons as compared with both potassium and ammonium salts.

The iodides and bromides of potassium and ammonium are so largely used that the importance of the foregoing is apparent if no further conclusion than this be drawn :— *that, the action on one tissue being selected and all other conditions being kept as far as possible identical, if one drug prove itself more active than another, it is at least not improbable that this same drug will also prove itself more active under the more complex conditions presented by the organism as a whole.*

A very guarded conclusion is obviously the only one that can be drawn when one passes as here from simple to very complex conditions, but the above conclusion is surely warranted.

Hence these experiments would suggest the substitution of the bromides and iodides of sodium for those of ammonium and potassium, and the use of those of ammonium preferably to those of potassium ; but the very wide gap separating sodium salts from both ammonium and potassium points especially to the use of the first, and the more so that, so far as clinical evidence goes, it is to the effect that, therapeutically, the salts of sodium and ammonium are as powerful as those of potassium.

In conclusion, one or two points may be touched upon. The very slight degree of poisonous action of the chloride, bromide, and iodide of sodium, was such that very large quantities of the drugs had to be added, so much so, that physical changes, such as osmosis, became probably important factors in the arrest of the heart. This, of course, would not affect the previous statements, but would rather

place still lower in the scale of poisonous action the above sodium salts, for clinically, the doses given would never even approach those here used, so that physical conditions would scarcely become factors in their therapeutic action.

These physical conditions may, however, account for the peculiar occurrence of piling up under the influence of continuous faradisation, which phenomenon was constant for the above sodium salts, and which Fig. VII illustrates.

It will be seen further on that with sodic citrate, of which salt considerably smaller quantities were used, this phenomenon did not appear.

Another point which touches a subject of very great importance may also be noticed. Looking over the results obtained, it will be seen that the chlorides, bromides, and iodides of potassium closely resemble each other in their action; the same is true of the ammonium salts. The sodium salts are best not considered here, since the physical elements introduced might obscure any such relation existing. The facts then to be put together are:—that salts resembling each other in having a common base resemble each other in their action.

The statement, that of a series of salts with the same base, the poisonous action is dependent on the base is not new, certainly with regard to potassium salts. These experiments, so far as they go, tend to confirm this statement, which, however, can scarcely represent the whole truth, for looking back at the table of quantities, it will be seen that the citrate of potash is as poisonous as the chloride, and rather more so than the iodide and bromide; it will be said, this is in direct opposition to clinical evidence.

It would be so, if one is to hold that the frog's heart as here used takes into account the whole action of the drug, but, if a salt do not act as a whole but individual elements composing it keep their identity, so far as action goes, then one must assume that one side or aspect of the drugs examined is here left out, and that the test is an imperfect one.

This again does not invalidate the conclusions drawn, for, given the bromide, say of three different bases, and that the basic element alone is taken account of, one must assume that the unaccounted-for bromine element being constant in all three, may, after the manner of all constants, be disregarded, and the salts be represented *relatively* in terms of their differences, *i.e.* of their bases.

The Citrate Group.

These organic salts were chosen in order to contrast them with the very definite group of the iodides, bromides, and chlorides amongst inorganic salts.

Citrates of soda, ammonia, and potash.

Of soda and potash the tribasic salts were employed, of ammonia the di-ammonic salt.

With reference to the quantities used, an important element is the water of crystallisation, which varies in the above three salts. Thus:

The molecule of tri-potassic citrate contains one molecule of water of crystallisation	$K_3C^6H_5O_7 + 1Aq.$	Mol. wt. = 324
That of tri-sodic citrate, 5·5 of water, the crystals having the composition .	$2Na_3C^6H_5O_7 + 11Aq.$	„ $= (357)_2$
That of di-ammonic citrate, none .	$(NH_4)_2HC^6H_5O_7$	„ = 226

The quantity of ammonic citrate which would contain the same number of atoms of the radical ammonium as the sodium and potassium salts, *i.e.* three, would be represented by the number 339. So that approximately equal weights of all three salts would contain the same number of atoms of potassium, sodium, ammonium.

Tri-sodic citrate.—This salt affects frequency but slightly, and so long as the contractions are visible, rhythm is manifest.

One case out of the six was somewhat exceptional, here

the effect on frequency was much more marked in the way of slowing, though even here in the final stages there reappeared a faint waviness of the trace, indicating faint spontaneous contractions.

The effect of continuous faradisation was certainly not diminished, if anything, it was increased. Even after the beats had completely ceased or had been reduced to a minimum, complete tetanus, though of a low altitude, was obtained.

In no case was the piling up obtained, which the chlorides, bromides, and iodides of sodium showed, when continuous faradisation was applied after the heart had been arrested by the drug.

The stress, as a rule, fell early on contractility, the contractions growing feeble rather rapidly, whilst rhythm suffered but slightly. In four out of the six cases, and doubtfully in a fifth, a certain amount of recovery took place under the action of the drug, *i.e.* the beats after reduction to a certain point, increased again slightly in spite of continued addition of the drug. The recovery was not, however, of long duration.

The early and sudden action on the height of the trace is noteworthy, in five out of the six cases it was marked, so that by far the larger amount of the drug was spent in destroying the small residuum of contractility remaining after the above primary effect.

The quantities employed were:

I.—Dec.	31.	Temp. of room	8°.	Quantity 16 c.c.
II.— „	31.	„	8·5°	„ 14
III.—Jan.	2.	„	11°	„ 12·5
IV.— „	2.	„	12·5°	„ 13
V.— „	6.	„	17°	„ 5
VI.— „	6.	„	17°	„ 5
				65·5

Average 10·83 c.c. = 1·083 grms. = 16·66 grains.

Citrate of ammonia $(NH_4)_2HC^6H_5O_7$.—In all six experiments spontaneous beats occurred up to the very end,

i.e. so long as contractility remained. As to quickening or slowing no constant effect was produced, but in two, out of the six, an effect similar to that caused by potash obtained, viz., a rather sudden slowing of the rhythm, with no appreciable change in contractility. As was the case with soda, a certain amount of recovery occurred after the beat had been primarily reduced.

With respect to faradic excitability, here, as with the soda salt, there was certainly no diminution, at any rate so long as the height of the beats were of sufficient value to give definite results in this direction. If anything, the change was towards increase.

When the contractility had been destroyed by the drug and single stimuli were without influence, continuous faradisation was equally without effect, *i.e.* no piling up obtained.

In four out of the six cases there was distinct primary increase in height of beat, in one it was doubtful, in one the only effect was a broadening of the beat. The stage of increase was of but short duration. The recovery before mentioned occurred in four out of the six cases; it took place after reduction of the beat to a very small quantity, and was but very slight. The decline in the height of the beats was gradual in some, in others occurred rather rapidly.

Quantities:

I.—Dec.	30.	Temp. of room	10°.	Quantity	2·4 cc.
II.— „	30.	„	10°	„	3·3
III.—Jan.	4.	„	15°	„	3
IV.— „	4.	„	16°	„	4
V.— „	5.	„	17°	„	3·9
VI.— „	5.	„	18°	„	3·4
					20·0

Average 3·3 c.c. = 0·33 grms. = 5·09 grains.

Citrate of potash.—The effect of this salt was primarily to slow the rhythm. This set in early, appearing as a rule after the first dose, though it often went on increasing with the first few doses. As a rule, this early effect was

without a corresponding effect on the height of the beat; thus the rate might be reduced by one half with little noticeable effect on the height of the trace.

Complete inhibition did occur in five out of the six cases, but not till the beat had been greatly reduced, viz. to $\frac{1}{4}$th, $\frac{1}{3}$th ($\frac{1}{3}$th—$\frac{1}{6}$th), $\frac{1}{7}$th, $\frac{1}{5}$th respectively, of the original height of the beat. This effect, then, was much less prominent than was the case with the chlorides, bromides, and iodides of potassium. The primary slowing above noted did not go on increasing at the same rate, and as a rule a stage followed in which great reduction in height occurred with scarcely appreciable change of rhythm.

Faradic excitability was markedly diminished, and a current giving strong tetanus before addition of the drug, finally failed to give any, or indeed only an initial contraction.

When the heart had been arrested by the drug and single shocks caused no effect, continuous faradisation was equally without effect.

There was no primary increase in the height of the beat, but decline from the very commencement; but whilst in the early stage the stress appeared to fall on rhythm—the decline in height being very slight—later on the effect on contractility became marked, the trace in some falling rapidly without corresponding effect on rhythm.

Quantities:

I.—Dec.	29.	Temp. of room	7°.	Quantity	0·9 c.c.
II.— „	29.	„	7·5°	„	1·8
III.—Jan.	3.	„	15°	„	1·8
IV.— „	3.	„	16°	„	0·9
V.— „	7.	„	14°	„	2·5
VI.— „	7.	„	14°	„	1·8
					9·7

Average 1·6 c.c. = 0·16 grm. = 2·46 grains.

The results obtained from the citrate group may be thus summarised:

Sodium and ammonium salts affect *excitability* but slightly; they arrest the heart by destroying *contractility*.

Both appear, if anything, to increase *faradic excitability*.

Specially it must be noted, that when the heart has been arrested by sodium citrate, continuous faradisation is without effect, *i.e.* no piling up obtains. And, further, that the sodium citrate is much more poisonous than the sodium salts of the chloride group.

With respect to ammonium citrate, note that a primary increase in the height and breadth of the trace is the rule.

Potassium citrate affects both *excitability* and *contractility*, but the effect on excitability is much less marked than is the case with the potassium salts of the chloride group; still, the effect was decided, especially in the end stages.

There is nothing special as to the action on contractility. Faradic excitability is diminished from the commencement.

Comparing these results with those of the chloride group, we note the agreement of the sodium and ammonium salts, in the slight effect on excitability, and in the mode in which they arrest the heart.

They contrast, however, in their effect on faradic excitability; thus, whilst the chloride group lessens, the citrates appear to increase this.

In respect of sodium citrate, the two points specially mentioned, viz. relatively small dose and absence of piling up go together, and are of importance in relation to the chloride group, since here we had large dosage, and in all cases the final mounting up of the trace under continuous faradisation. The citrate results are in conformity with the view that this mounting or piling up indicates physical change in the muscular tissue.

In addition to the agreement above noted in respect of the effect on excitability and contractility, the ammonium citrate agrees with the ammonium salts of the chloride group in its primary effect of increase in height and breadth of trace. The only point of contrast hence is in respect of faradic excitability.

The potassium citrate contrasts with the potassium salts of the chloride group in its much slighter action on *excita-*

bility. It is true complete inhibition did obtain in five out of the six cases, but the beat was greatly reduced before such occurred.

In its influence on faradic excitability, there is agreement with the chloride group.

The doses required to destroy contractility are for both the ammonium and potassium citrates about the same as required in the chloride group. This question of poisonous action has been discussed with reference to clinical experience.

In the case of the salts of potassium we have the most complete series. Thus we have examined the hydrate, the chloride, bromide, iodide, and the citrate.

Taking the hydrate as the starting point, we note here—

1st. The tendency to produce *persistent spasm*.

2nd. The tendency to produce *inhibition*.

3rd. The tendency to lessen *faradic excitability*.

Passing to the chloride group, we note that the tendency to produce persistent spasm has disappeared, that the action on spontaneous excitability is, however, still very marked, also that on faradic excitability.

Passing to the citrate of potassium, we note the absence of any tendency to produce persistent spasm, that the action on spontaneous excitability, *i.e.* the tendency to inhibit, is much diminished; that the action on faradic excitability still remains, though it is difficult to state comparatively the degree in which it persists.

With the exception, then, of the persistent spasm feature, we note a similarity in the nature of the action running through all these salts of potassium.

The ammonium series is less complete since the influence of the hydrate on the spontaneous working of the heart could not be so completely tested.

Here we note, however, that the tendency to produce persistent spasm runs through all the salts of ammonium; though in the chloride group and in the case of the citrate, this tendency is but slight, appearing early and for only a short period.

Further, amongst the salts of ammonium there is agreement in the negative quality of slight action on rhythm.

As to the effect on faradic excitability there is no agreement, for whilst ammonia increased the above the iodide lessened it, whilst the citrate increased it. (The chloride and bromide were not examined.)

The sodium salts can be less easily compared, for in the chloride group, physical action probably comes into play. The persistent spasm which the hydrate excites does not appear with any of the salts. The action on spontaneous excitability is slight as compared with potash salts, and in this respect there is agreement throughout the sodium salts. The action is, however, more marked than in the case of ammonium salts. As to the effect on faradic excitability, the sodium salts show as little constancy of action as those of ammonium; thus, whilst soda increases faradic excitability the iodide lessens it, whilst the citrate shows, if anything, a tendency towards increase.

EXPLANATION OF DIAGRAMS.

Diagram 1.

Fig. I.—Illustrating "genesis of tetanus" by fusion of beats, the result of continuous faradisation.

A, B, C, show the effect of increasing the strength of the current, the frequency of interruption of the primary current remaining the same.

The figures above the lines represent the positions of the secondary coil.

D, represents a later stage.

As the strength increases from 7·5—7—6, it will be seen that the more complete tetanus also corresponds with the greater number of contractions. In D the fusion is so complete that individual beats are no longer countable.

FIG. II.—Shows effect of sodium hydrate on the excitability of the ventricle to continuous faradisation.

Nov. 25. Temperature of room 16° C.

A. Before the addition of sodic hydrate.

B. After 25 minims of a 1 per-cent. solution.

C. After 75 „ „ „

FIG. III.—Shows similarly the effect of ammonium hydrate.

Nov. 26. Temperature of room 15·5° C.

A. Before the addition of ammonium hydrate.
B. After the addition of 10 minims of 1 per-cent. solution.
} see Diagram 2.

C. „ „ 24 „

D. „ „ 55 „

Fifty-six minutes from first dose.

DIAGRAM 2.

FIG. III, A. and B. (see above).

FIG. IV.—Shows effect of potassium hydrate. A 10 per-cent. solution was here used.

Feb. 12. Temperature of room 16° C.

A. Before addition.

B. 10 minutes later after 0·15 c.c. = 2·55 minims. The tetanus is here taken in the earliest stage of the potash effect, note the faint evidence of diastolic rise.

C. 10 minutes from B, after 6 minims in all. Note the diminution of effect, together with the presence of considerable amount of persistent spasm.

D. and E. are after 7·6 minims and 9·3 minims respectively, they show progressive decrease in excitability.

FIG. V.—Jan. 28. Temperature, 16·5° C. Bromide of ammonium, 10 per-cent. Chosen as typical of the action of ammonium salts of this group, viz. chlorides, bromides, iodides.

A. Shows effect of first addition of 0·2 c.c. = 3·4 minims also a few beats before the addition.

B. About five minutes from first addition; 0·6 c.c. = 10 minims have been added.

C. 10 minutes from B, 1·8 c.c. = 30·5 minims have been added.

D. 2 minutes from C, 2·2 c.c. = 37·3 minims „

DIAGRAM 3.

E. Comes immediately after D, 2·4 c.c. = 40·6 minims was the total quantity added.

Note certain amount of persistent spasm in A and B, the beats are too frequent for diastolic contraction to appear between the individual contractions.

Note slight irregularity preceding the end stage.

FIG. VI.—Jan. 19. Temperature 15° C. Bromide of potassium, 10 per cent.

A. First dose 0·1 c.c. = 1·7 minims.

B. 4—5 minutes later, after 0·3 c.c. = 5·07 minims

C. 2—3 minutes later, fifth dose = 0·5 c.c. = 8·45 minims

D. 3 minutes later. The last spontaneous beat is here represented, viz. after 0·8 c.c. = 13·52 minims, so that hence on there was complete inhibition.

E. 10 minutes later, 1·1 c.c. and 1·2 c.c. (18·6 and 20·3 minims) = the quantities added. The beats are the result of excitation.

F. 8—9 minutes later, after 1·4 c.c. = 23·7 minims.

G. 5 minutes later, final stage, 1·7 c.c. = 28·8 minims, the total quantity added.

Note the primary slowing in B and C, the rhythm was further somewhat irregular; in D, just before complete inhibition, there was considerable irregularity. The subsequent charts show that, though inhibited, contractility still remained, the figures above the beats represent the positions of the secondary coil.

FIG. VII.—Showing effect of continuous faradisation after heart has been arrested by bromide of sodium. The charts are typical of the chlorides, bromides, and iodides alone.

A. Jan. 24. Temperature 17·5° C. Sodium bromide 10 per-cent. The effect of continuous faradisation with the secondary coil at 4, and at 0 (pushed home) is shown. The previous want of effect of single shocks is shown, and of continuous faradisation at 6.

B. Jan. 27. Temperature 17° C. Sodium bromide 10 per-cent. Similar to above, this perhaps represents the more usual degree of effect produced.

Diagram 1.

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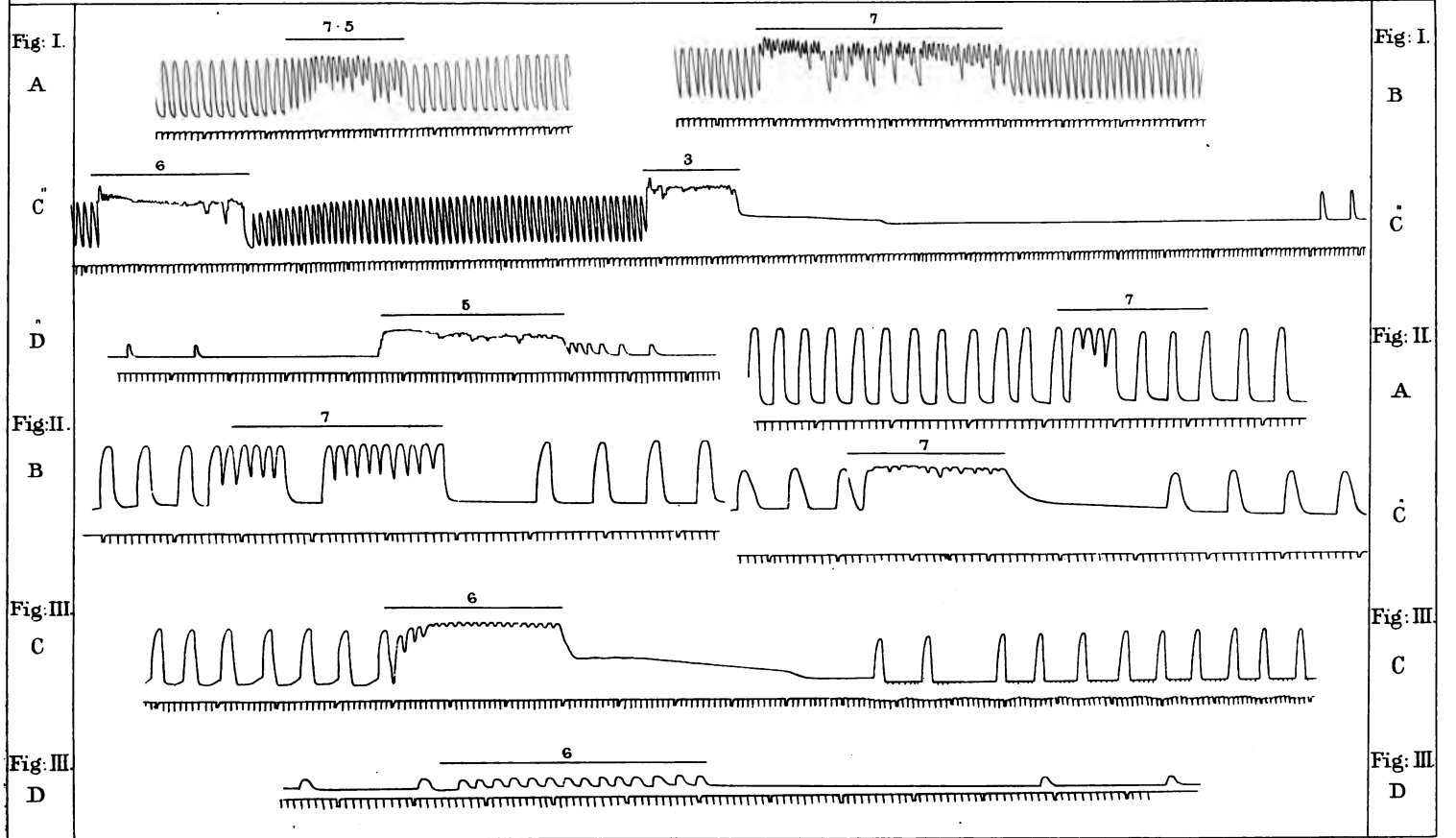

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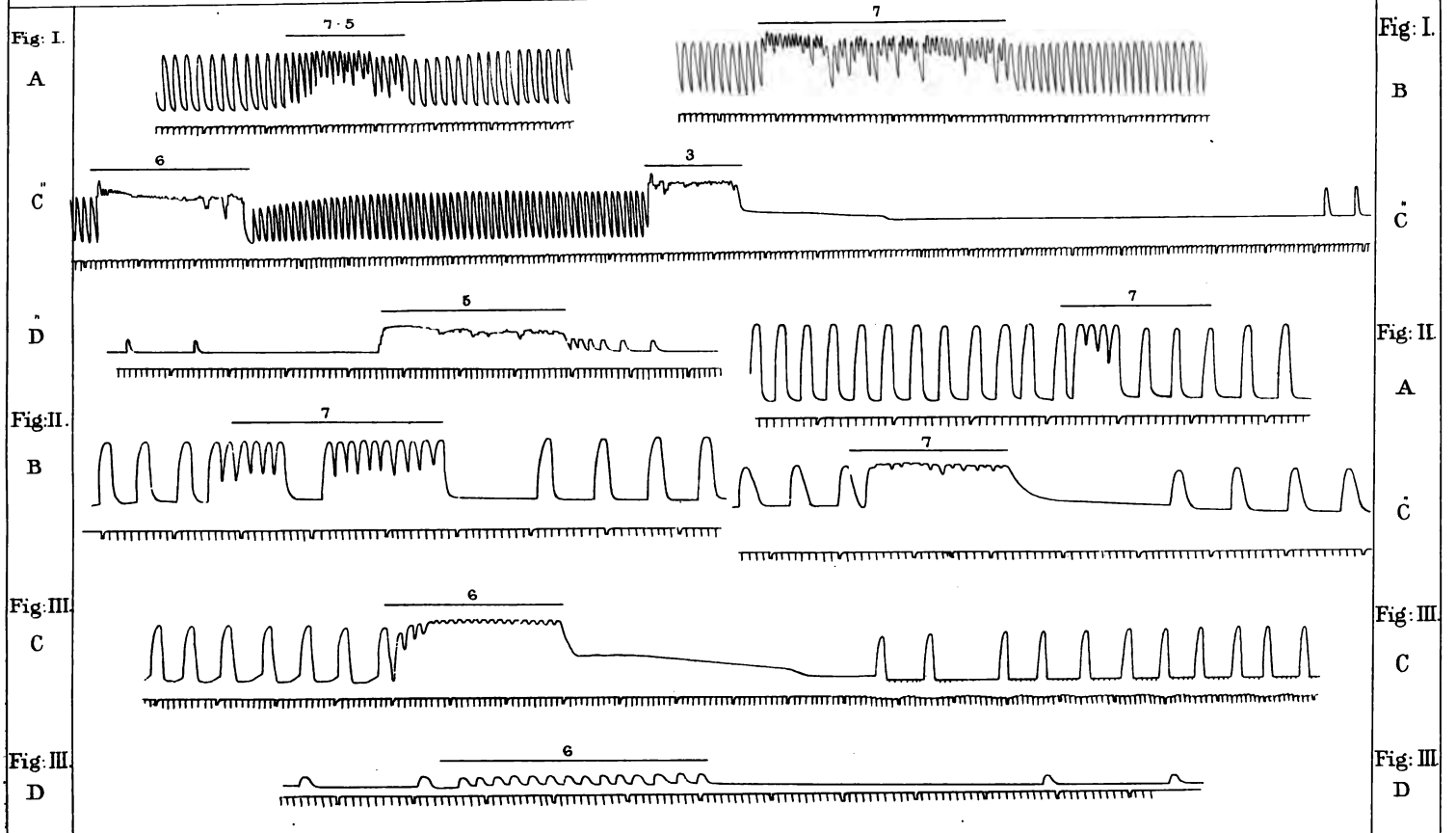
Diagram 1.



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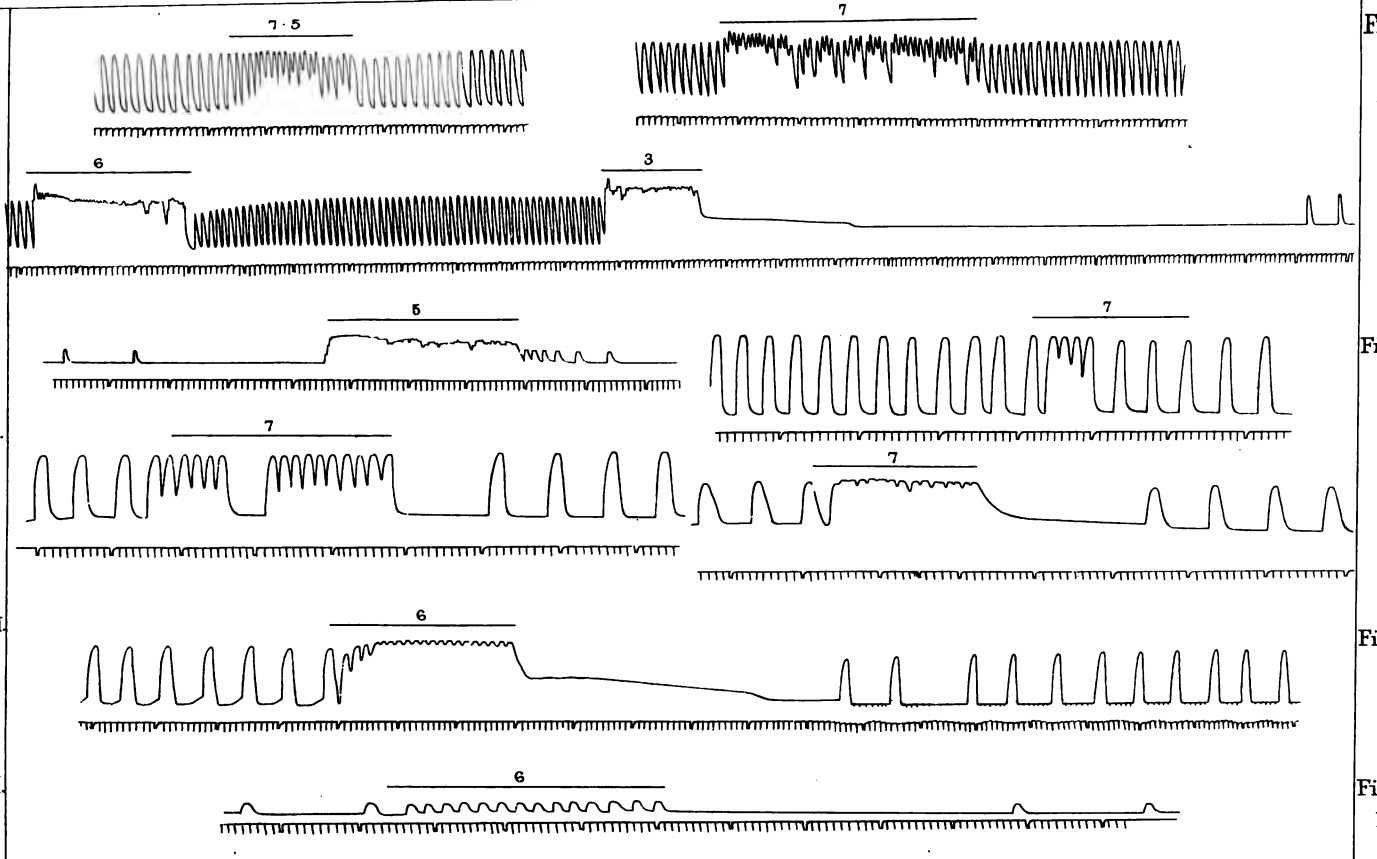

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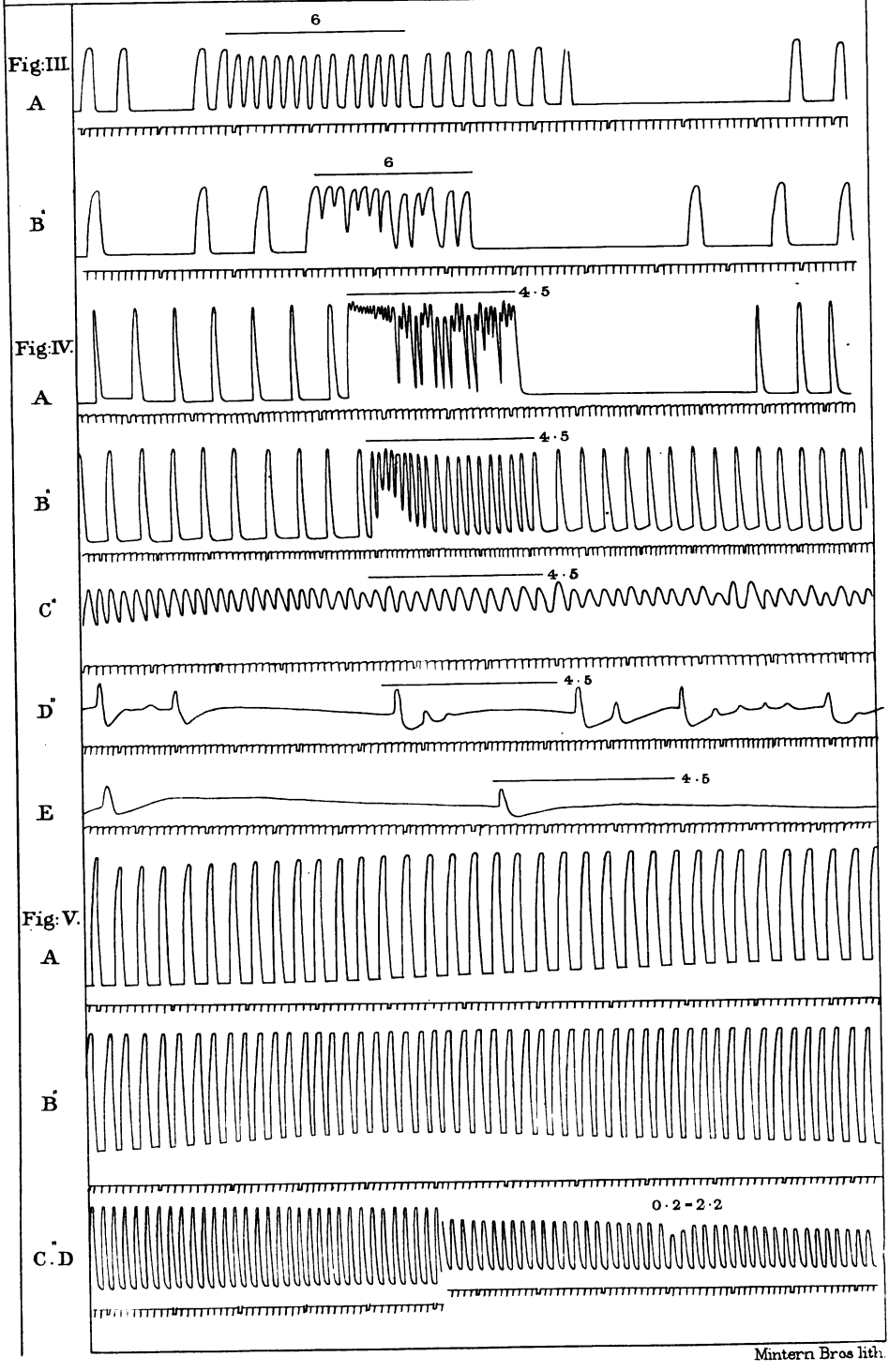

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Diagram 2.

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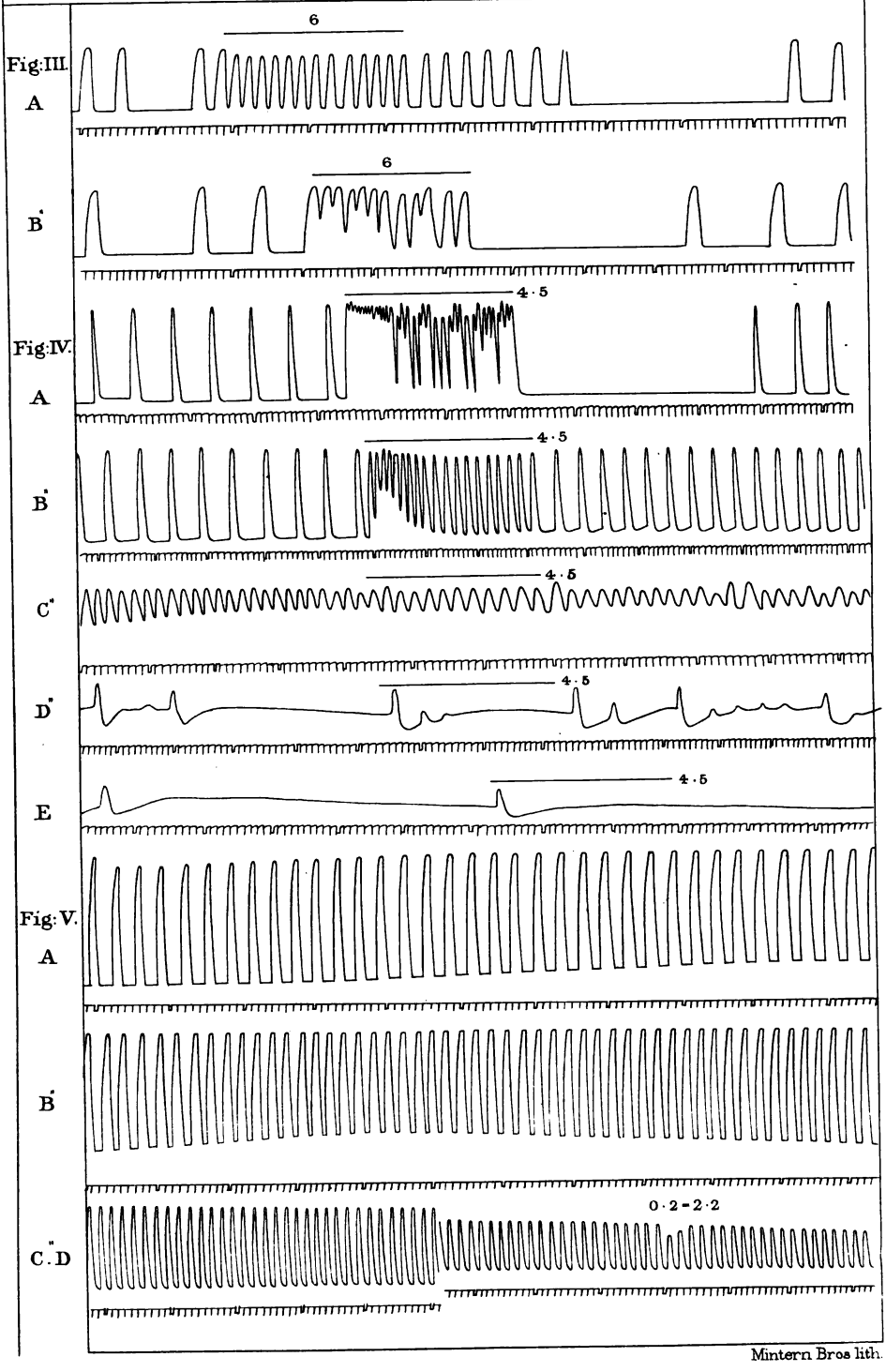

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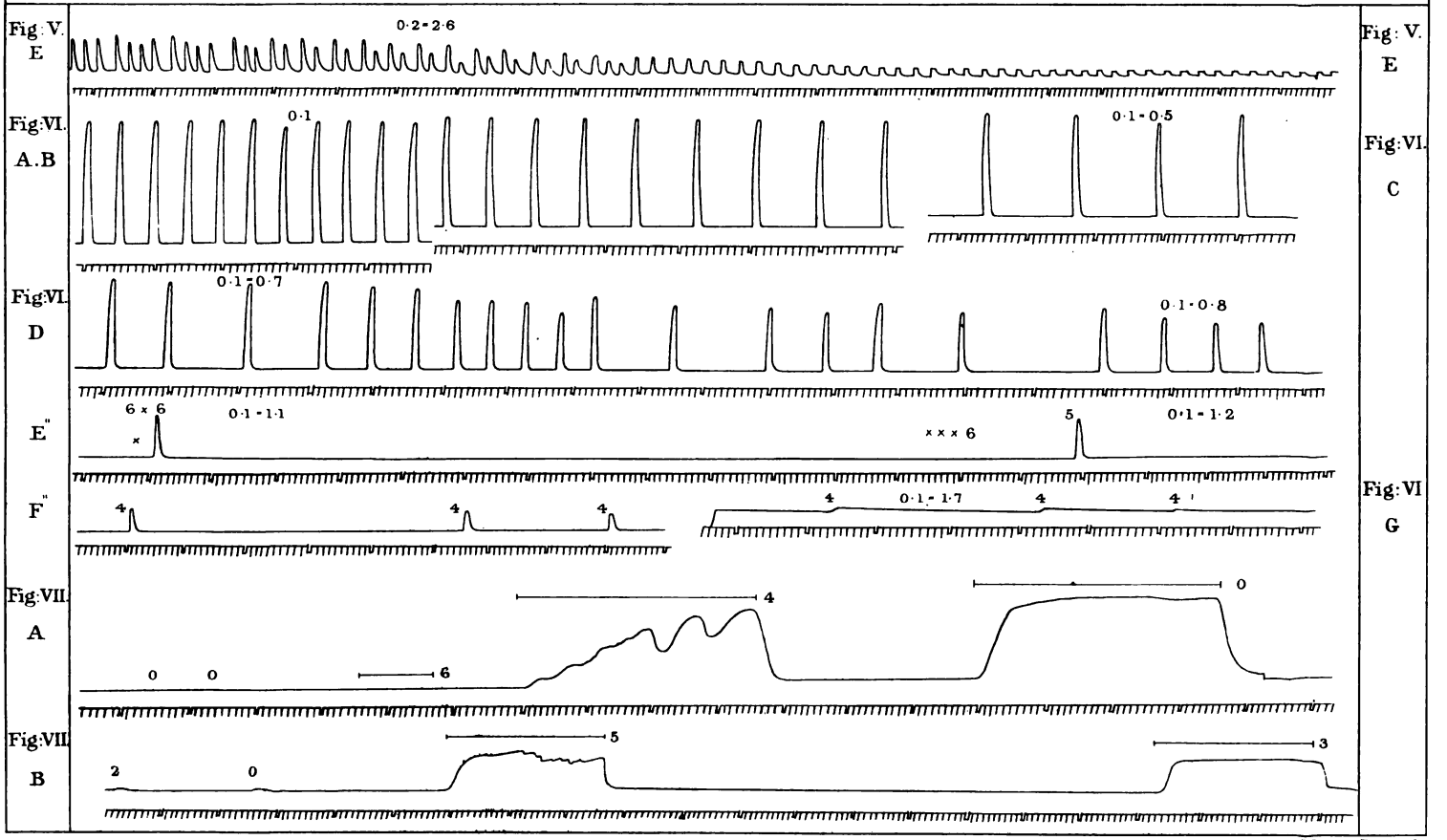

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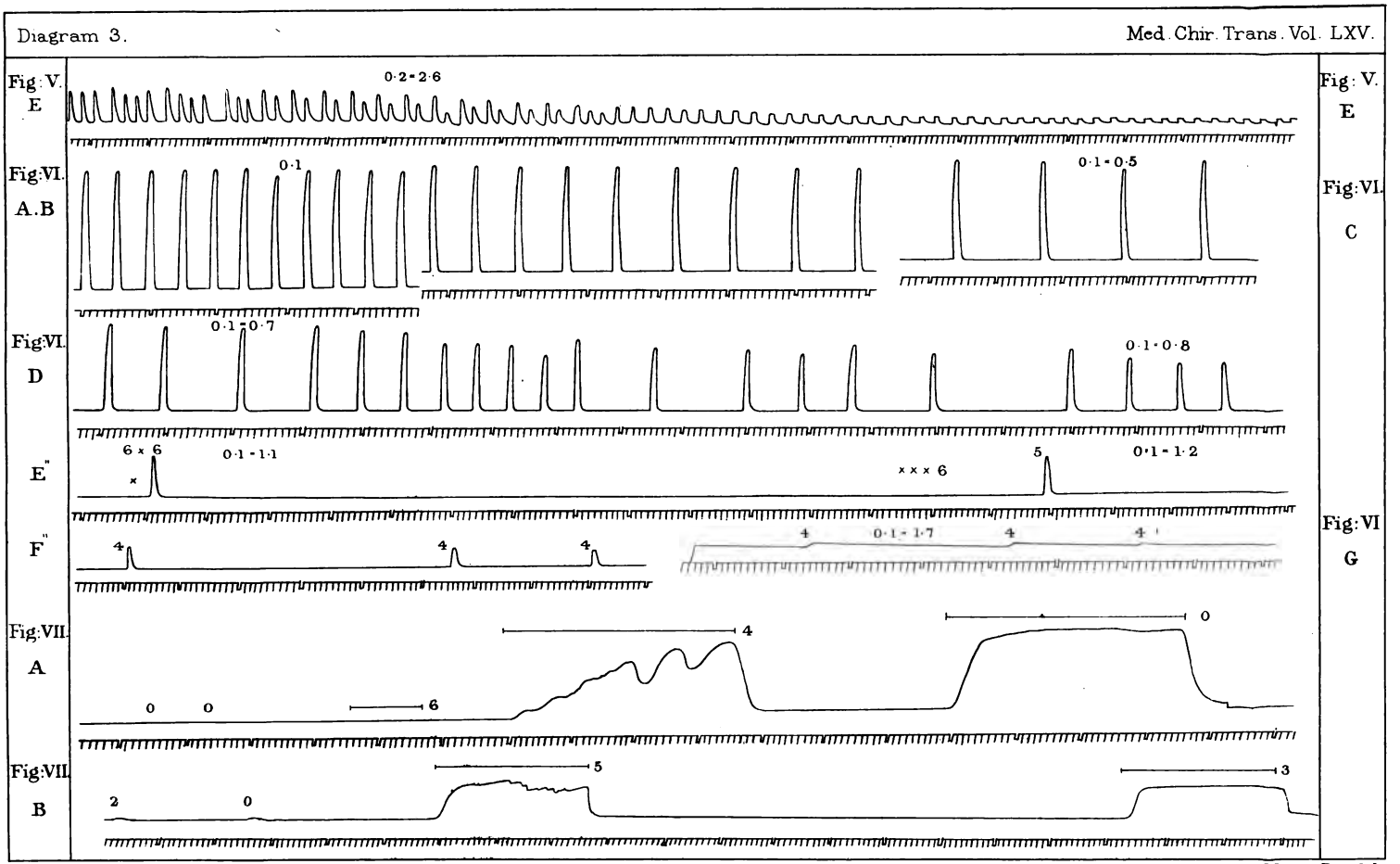

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ADDITIONAL

CASES OF OSTEITIS DEFORMANS.

BY

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(Received April 17th—Read June 13th, 1882.)

I SHOULD not have offered to the Royal Medical and Chirurgical Society a mere collection of cases such as this paper contains, if it were not for the hope that they may help to clearly indicate the chief characters of the disease to which I venture to give the name of osteitis deformans, and which, so far as I know, was first described in the paper published in the 60th volume of the Society's 'Transactions.'[1] Since that time, about five years ago,

[1] After the publication of the paper I found that the name ostitis deformans had been given by Prof. Czerny, of Freiburg, to a disease described by him in the 'Wiener medizinische Wochenschrift,' September 27th, 1873. It is mainly, as he says, "Eine lokale Malacie des Unterschenkels," a rather acute inflammation of the lower part of the tibia and fibula, inducing softening and angular bending, and then followed by hardening. A specimen of the only instance of this disease that I have seen has lately been presented to the Museum of the College of Surgeons by Dr. Butt, of Hereford. Like all Prof. Czerny's cases it occurred in a young man.

It may be well to add also that the sternum, a clavicle, and a rib of the man, whose case by Saucerotte is referred to in my last paper, are in the Musée Dupuytren. They were obtained by Saucerotte many years after his account of the case was published. The description of them in the 'Dupuytren Catalogue,' vol. ii, p. 148, leaves it very doubtful whether the disease was osteitis deformans.

I have seen seven cases of the disease, and have recorded concerning them the facts which follow :

Case 1.—May 29th, 1878. A lady, æt. 65, having no appearance of general ill-health and looking her age, complained chiefly of what she deemed to be rheumatic and neuralgic pains of her back and lower limbs. She ascribed them to exposure to cold thirteen years ago ; for she had rarely since that time been free from pain, and had lost strength and health ; and in the last year or more, had suffered with what she considered to be attacks of bronchitis and asthma.

Soon after the beginning of her pain, that is, about ten or twelve years ago, her daughters thought that she was losing in height, and that the shape of her head was changing ; and from that time she had been becoming less tall, till now she had lost four inches and a half in height, and stooped low with her head forward and her chin raised.

She had marks of slight gouty affections in some knotted knuckles, and frequent flatulence and occasional excess of lithic acid in the urine. But she had never had fever, ague, or any acute illness, and had borne five children in rather hard labours, without ill consequences.

Her father had been gouty and died in old age ; her mother died young after parturition. She did not know of any case of scrofula, consumption, or cancer having occurred in her family.

Her head, though she said it had always been a remarkably large one, was certainly enlarged, and chiefly by convex bossed additions over and about the junction of the frontal and sagittal sutures and above the temporal sutures. They were symmetrical, and might be guessed to be additions of one third or half an inch in thickness, perhaps additions to a general thickening of the cranium. But they neither were nor had been associated with headache or any other local trouble, and sight, hearing, and the other senses were unimpaired.

The dorsal spine was curved with a very marked posterior rounded curve, inclining a little to the right, without any compensating curve to the left below it. The curve produced a low stooping posture, with very prominent right shoulder, and might be estimated as shortening the trunk about two or two and a half inches.

The ribs were nearly horizontal, flattened at the sides, and, even in deep inspirations, nearly motionless. The respirations were almost wholly diaphragmatic, with elevation of the sternum. They appeared to be sufficient during quietude, but in any hurry or mental emotion, or any unusual exertion, great distress of breathing was felt, and walking upstairs seemed even dangerous; she was always carried up.

The lumbar spine appeared of natural form, so did the pelvis, and likewise all parts of the upper extremities.

The femora were exceedingly curved outwards and forwards; the left rather more than the right. Their shafts felt in their whole length, especially, I think, in their lower half, large, rounded, thickened.

Similarly the tibiæ were curved forwards and were very large in their whole length. Their anterior surfaces felt nearly twice as wide as in nature, smooth, and with large rounded margins.

The feet and all the articulations of the lower limbs appeared quite healthy. None of the enlarged bones were tender on pressure.

The likeness of the forms of the trunk and lower limbs in this case, and in the first case recorded in my former paper, was very striking. The similarity of disease could not be doubted.

The patient lived two years and a half after this note of her case was made, and during this time was under the care of Mr. Haynes, of Stansted, to whom I am indebted for being able to report that little change ensued in the bones of the lower extremities; that the skull became more deformed, especially with a broad, high boss along its upper middle line; and the spine more curved

and prominent in its dorsal part. Death ensued in consequence of Bright's disease and valvular disease of the heart, with extreme anasarca. It did not appear due in any degree to the disease of the bones, unless it were that the difficulty of breathing was aggravated by the deformity of the chest. There was no indication of cancerous disease of any part.

Examination after death was not allowed.

Case 2.—February 17th, 1879. A man, æt. 62, looking in everything but shape completely healthy, and feeling well and fit for work, even more fit than ever for any mental work, told that he had been healthy all his life, and had been six feet and a half inch high, slim, well made, and active, a volunteer, and given to all kinds of exercise. He was not aware of any unhealthy inheritance, certainly not of gout. He did not know the very beginning of his illness, or any cause for it. He first observed, distinctly, that his stature was less, and that his left limb was rather stiff and lame, in May, 1871, when he was 54. A bone-setter then told him that his hip was "out" and professed to reduce it, and later in the year he was treated for chronic rheumatism at Buxton and obtained there some relief from pain. From this time he appeared to be constantly losing height and becoming more misshapen; but he had never suffered more than might be ascribed to chronic rheumatism of moderate severity in the spine and limbs, and had never left off work or the taking of moderate exercise. He had been obliged to wear larger hats than he had been accustomed to, and had twice or more in the last two years increased the size, but he never had a headache of any kind. Once only in the eight years he had been ill. This was with jaundice, of no great severity, in 1876, and once or twice he had had dyspepsia. Lately he had become rather deaf.

Now he was five feet eight inches, having lost four inches and a half in the eight years. His head was large and well formed, but disproportionate to his face, and his

frontal veins looked very full and tortuous. The face was natural. His neck was very short and stiff, and the head was habitually held forward with the chin rather in advance of the sternum, and not more than two inches above it when the face was directed straight forward. The spine was very short, nearly straight, as if its anterior and posterior curves were straightened in the shortening, and it was far too little flexible. The ribs were crowded, nearly horizontal, and hardly raised in inspiration. His in-breathing was almost wholly with the diaphragm, and, when deep, with great uplifting of the shoulders. Yet he called it "pleasant breathing," and was never troubled with it unless on fast walking. The abdomen was short, prominent, overhanging, deeply folded. The clavicles were very big and full-curved, twice as big (at a guess) as they should have been. The arms and hands appeared quite natural, muscular, and agile, with all the power of writing, fencing, and other uses that they had ever had. I observed nothing wrong in the pelvis. The femora were big and very curved; the left, which had been longer and more affected than the right, was curved outwards and forwards, the right forwards and but little outwards. The tibiæ were big in their whole length, broadly rounded, very curved forward. The feet and all the joints of the lower limbs appeared quite healthy, but the mobility of the hips and knees was lessened.

This patient still lives in fair general health, but with all his deformities increased.

Case 3.—April 12th, 1879. Mr. M—, æt. 66, formerly a colonial judge, looking older than his years, and dry and rather shrivelled, had had generally good health, but once had fever, and once, about fourteen years ago, congestion of the brain. He had been liable to general rheumatic pains, but not more in the lower limbs than in other parts, and he came for advice about his left ankle, which had become less movable in consequence of the

great overhanging of the tibia and the very acute angle in which he habitually held it whilst standing.

His appearance and posture were very characteristic—with his head forward, his shoulders raised, short trunk, legs apart, feet turned out—the right foot forward, the left behind—and both legs greatly curved. Except that he was a shorter and smaller man, the photographs published in the 60th volume of the 'Transactions' might have suited him.

In ten years his height had diminished about two inches, chiefly, I think, from shortening of the spine. The head was well formed and large—it always had been so—and he had always had difficulty in finding a hat large enough for him; but the only indication of increase in it which he had observed was that his present hat was rather tight. The enlargement, however, if any, must have been very little. I could not find any sign of enlargement in the upper limbs. The ribs were nearly horizontal, and less mobile than they should have been. There was less than a finger's breadth between the lowest rib and the crest of the ilium. The spine was not curved below the lower part of the neck. Each femur in its lower and middle thirds felt thick, and curved forwards and outwards, the right more than the left; the necks of the femora were lowered. The tibiæ were in their whole length very big, rounded at their edges, and exceedingly curved forwards; they felt uneven and knotted, and the integuments in front of them were over-hot; they overhung the feet, and in standing the legs and feet were at acute angles.

The date of the beginning of these changes was uncertain. They had, probably, been in very slow progress for not less than ten years, but they had given so little trouble that they had not been closely watched. No cause of them could be told. One brother was gouty, but the patient himself had not had any marked gout, and had lived a simple temperate life. The disease probably began in Melbourne.

Case 4.—1880. Mr. S— was 51 years old, a very

small man, who had never weighed so much as eight stone, and now was about seven stone eight pounds.

He had so exactly the characteristic form that one could instantly feel sure of the diagnosis of his case. His head was inclined forwards and downwards, his neck looked short, his back was curved backwards, his chest low and small; his pelvis looked broad and womanly; his arms hung low. The lower limbs were arched and divergent: the right advanced, the left as if dropping back, the left foot even more than the right, flat and overhung. The head was, he believed, quite unchanged in size and shape; it was long and flat-sided, but he had not had occasion to change his hat. Both clavicles were enlarged, they felt thick, clumsy, rough. The ribs were very little mobile; the chest was lifted as in one piece when he drew his breath deeply, raising his shoulders. The chest was "square," the ribs flattened at the sides, and bending round with an angle to form the similarly flattened chest front. The ribs severally did not feel as if enlarged. The liver appeared to be pressed high up into the chest; there was dulness to the nipple. The abdomen was wrinkled and undulated as if its walls were too long. The spine was deeply and roundly curved in its dorsal part, but straight in its lumbar. The femora and tibiæ were very large, especially the lower halves of both, and all were very curved; the femora forwards and outwards, the tibiæ more forwards. The joints felt and looked healthy, but the left knee-joint was painful in movement and rather stiff. Except that he was very thin and dry, all the structures except the bones appeared quite healthy. He could find nothing for complaint but the pain in his knee, which had now lasted some weeks.

He ascribed his illness to a walk down Vesuvius ten years ago. He was very tired with it, was laid up with pains in his lower limbs, and from that time, as he believed, his legs began to curve. Since that time he had not had any considerable illness, but from time to time pains of no great severity confined to the lower limbs. Many years

ago he had had syphilis, and there were two scars of tertiary sores on the lower limbs.

I advised him to take iodide of potassium, but it did no good. He remains in good general health and with very slowly progressive changes in his limbs.

In the fifth case, which so far as the notes described, resembled the preceding, Mr. Butlin examined one of the tibiæ post mortem. The condition differed in no degree from that of the bones described in the 60th volume of our 'Transactions.'

Case 5.—January, 1882. I have so few notes of this next case that I should not have inserted it but for the examination of a portion of one of the diseased tibiæ which Mr. Butlin was so good as to make.

The Rev. Mr. G— had been an active, healthy man, till he was nearly sixty, and he died at sixty-six; his chief signs of illness during this interval having indicated various attacks of irregular gout with bronchitis, emphysema, and diseased heart-valves. He was born of long-lived parents, and had no sign of general disease but such as might be ascribed to gout.

Indications of osteitis deformans had appeared about four years before his death and had slowly increased. The tibiæ especially had gradually become curved forwards, large, rounded, and uneven, and at first had been painful and hot. The femora were also slightly affected, and the spine had become roundly arched and over-hanging at its upper part.

Mr. Butlin reported that the portion of tibia which he minutely examined did not differ in any respect, so far as he could see, from the diseased bones of which his descriptions are recorded in the first case related in my former paper (see vol. lx., p. 46).

Case 6.—March 9th, 1882. Mrs. S—, æt. 62, mother of three children, and generally healthy, though with inheritance of gout, and having often flatulence and various

gouty symptoms. She was very thin, and her large bossed head, low stooping, and oblique posture, and separated lower limbs, with one far in advance of the other, might at once have sufficed for diagnosis. She believed that her disease began at least ten years ago, when first she observed some enlargement of the external angular processes of the frontal bone. From that time she had had various pains regarded as rheumatic or gouty in her back and lower limbs, and had observed her head growing larger, her spine curving, her height diminishing, her lower limbs bending and becoming weaker, till now she was nearly four inches shorter than she used to be, could hardly stand without support, and walked slowly and as if her right leg was too long.

Her head appeared enlarged at every part, and measured twenty-four inches in its largest circumference; it had a square look, and was bossed, chiefly over the frontal and parietal protuberances and over the lower part of the occiput. The orbital arches also appeared large and prominent, but in the bones of the face there was no apparent change. The dorsal part of the spine was very curved backwards and to the right, the chest was shortened, the ribs crowded but fairly movable. The pelvis seemed not changed. The clavicles were very large and curved, like those of a strong man, contrasting strangely with the thin weak look of the upper limbs, whose bones were slim and naturally straight.

Both femora and both tibiæ were very curved forwards and outwards, and felt large and rugged. Especially the lower parts of the tibiæ were enlarged, and there was little diminution of size between the shafts and the articular ends, which seemed as if over-hanging the ankles. Both shins felt hot.

The bones of both hands and feet were healthy unless for some nodular enlargement of the phalanges such as one sees due to gout.

The patient was not aware of any inherited liability except that of gout, or of anything that could have induced

the disease of bones. She had never been dangerously ill.

Lately her sight had become much impaired and still more lately she had been becoming deaf.

CASE 7.—May, 1882. In this case, a lady, æt. 58, presented the usual characters of the disease in a well-marked form, but had observed the first signs of it when she was only 28. At that time, during the fatigue of long nursing a relative, she had pains in her lower limbs and found her tibiæ becoming large and curved. Her spine also at this time began to curve. The disease was very slowly and almost painlessly progressive; its chief or only seats being the tibiæ, femora, spine, and clavicles; but when she was 53 she observed enlargement and slight curvature in the lower thirds of both radii. She ascribed this to the exertions she used in frequently lifting her invalid husband, and the morbid changes had continued slowly increasing ever since. Her head was very large, measuring twenty-four inches and a half in its greatest circumference, and looking as if unnaturally prominent in the middle and upper frontal part, but she was sure that this was only its natural size and shape.

With the exception of the disease of the bones and the consequent great difficulty of walking, this lady seemed in excellent health. She had lived very actively; had had two children without any ill consequences, and knew of no inherited tendency to disease unless it were to gout. She had had various treatment with both baths and medicines, but none had seemed useful even for a time.

The seven cases now related seem sufficient when added to the five recorded in the 60th volume, to justify the giving of a distinctive name and a definite general description of the disease observed in them. It usually affects many bones, most frequently the long bones of the lower extremities, the clavicles, and the vault of the skull. The affected bones become enlarged and heavy, but with

such weakening of their structure that those which have to carry weight or to bear much muscular traction become unnaturally curved and misshapen. The disease is very slowly progressive, and is felt only in pain, like that of rheumatism or neuralgia, in the affected limbs, and in increased heat at the tibiæ. But neither the pain nor the heat are constant, nor do they continue during the whole progress of the disease; and pain has not been observed in the head even in the cases in which the skull was very thickened. There is not any clear evidence of general disturbance of health. In all the cases traced to the end of life, death has ensued through some coincident, not evidently associated, disease, which has been aggravated by the condition of the bones only in so far as they may have diminished the range of breathing and the general muscular activity.

At present, with the exception of the seventh case, this disease has been observed as beginning only in persons over forty years old, and it has appeared in no usual relation, whether by inheritance or coincidence, with any other disease except gout. I have not found cases to be added to those mentioned in my last paper in which it was associated with cancer.

In all the cases I have seen, the general appearance, postures, and movements of the patients have been so alike that these alone might often suffice for diagnosis of the disease. The most characteristic are the loss of height indicated by the low position of the hands when the arms are hanging down; the low stooping, with very round shoulders and the head far forwards, and with the chin raised as if to clear the upper edge of the sternum; the chest sunken towards the pelvis, the abdomen pendulous; the curved lower limbs, held apart and usually with one advanced in front of the other, and both with knees slightly bent; the ankles overhung by the legs, and the toes turned out. The enlarged cranium, square looking or bossed, may add distinctiveness to these characters, and they are completed in the slow and awkward gait of

the patients, and in the shallow costal breathing, compensated by wide movements of the diaphragm and abdominal wall, and in deep breathing by the uplifted shoulders.

I have seen no case in which these characters are imitated except those of ankylosis of the vertebræ and ribs, to which I referred in my last paper, and which have been described by Dr. Allen Sturge[1] under the name of spondylitis deformans; but these are easily distinguished by the lower limbs being naturally straight and the clavicles and skull unchanged.

[1] 'Trans. of the Clinical Society,' xii, p. 204, 1879.

NOTES

OF

TWO CASES OF MALIGNANT PUSTULE,

TOGETHER WITH

A TABLE OF SEVENTEEN CASES TREATED AT GUY'S HOSPITAL.

BY

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WITH

A REPORT ON THE MICROSCOPICAL EXAMINATION OF SECTIONS OF SKIN AFFECTED WITH MALIGNANT PUSTULE, REMOVED DURING LIFE.

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(Received May 9th—Read June 13th, 1882.)

THE affection known as malignant pustule or charbon appears to be rare in this country. The writers of our surgical text-books speak of it as a condition of which they have had little or no personal experience. Since the account of this disease, which was published by Dr. William Budd, of Bristol, nearly twenty years ago, very little had been heard of it on this side of the Channel until the recent outbreak of what was termed "wool-sorters' disease" in Bradford. At Guy's Hospital, however, we have been aware, for the last nine years at least,

of the not infrequent appearance of this dangerous malady in our neighbourhood. This is, no doubt, due to the foreign fleeces and hides which are used in the great leather manufactories of Bermondsey. I find that since the autumn of 1873 at least seventeen cases have been admitted into the hospital. Three of these were recorded by me in the 'British Medical Journal' (vol. i, 1878), and another was published by Mr. Golding Bird in the 'Guy's Hospital Reports' (series 3, vol. xxiii). Last year two well-marked examples were admitted into my wards, as well as three others under the care of my colleagues. I have therefore thought that it would not be unprofitable to bring before the Society the details of my two cases, together with a table containing the chief facts relating to the seventeen cases which have occurred under my colleagues and myself. It appears to me of much importance to call attention to the frequency of the disease in connection with the leather trade, and to the facility with which it may often be cured even when the local and constitutional symptoms have been allowed to attain a considerable development. From occasional notices in our journals and elsewhere I am afraid that medical men sometimes fail to recognise it, and even after the diagnosis has become no longer doubtful that they do not appreciate the necessity of early operative treatment.

Case 1.—Frederick R—, æt. 31, who works at a hide warehouse, was admitted into Guy's Hospital on April 16th, 1881.

For eight days previous to the appearance of the pustule he had been working among Australian fleeces, and had not handled any of the hides, though he had been through the rooms where they were stowed. On Sunday, April 10th, he noticed a very small red spot on the right lower eyelid at the outer corner. The next morning the lid was puffed, and the spot had increased in size. He went to work feeling quite well, and he was rallied by his

companions on account of his black eye. On Tuesday the manager sent him to Guy's Hospital, where he was recommended to poultice the eye. On Thursday he came again, and the swelling was scarified. This day he felt poorly. He had lost his appetite, and was feverish. That night and the next he could not sleep. On Friday he was trembling very much, but the pain was not great, not as much as would have been caused by an ordinary boil. On Saturday he was admitted. His right eye was then completely closed; the lower eyelid was much swollen, and measured 2½ by 1½ inches. A considerable portion of its surface was occupied by an "ugly yellow vesiculo-pustular mass." The cheek and surrounding parts were greatly inflamed, especially the upper eyelid. The glands below the jaw were much enlarged. He was not in pain, but felt very miserable, and did not care what was done to him. I saw him in the afternoon. Upon his lower eyelid was the characteristic vesicular eschar, of which I shall give a more detailed description in the second of my cases, and there was much surrounding œdema. About 5 p.m. ether was administered, and the lower eyelid was removed by Mr. Jacobson down to the deeper strata of the orbicularis palpebrum. The muscular fibres had lost their natural colour, the superficial layer being green and sloughing, the deeper dark black, except towards the inner canthus. Numerous plugged vessels showed as scattered black points throughout, and there was enormous ecchymosis of the palpebral conjunctiva. The eyeball itself was healthy. Some incisions were made in the upper eyelid, and chloride of zinc applied to the surface left by the removal of the lower lid. Bacilli were found in the blood. There was no enlargement of the veins. Immediate relief followed the operation, and in five weeks the patient went out quite well, with the exception of considerable eversion of the lower eyelid, and a granulating wound which had not quite healed.

I am indebted to my colleague Mr. Jacobson, who

kindly operated on this case during my absence, and subsequently took care of it, for the permission to bring it before you, and for most of the details in the report.

Case 2.—Thomas W—, æt. 39, a tanner, was admitted July 11th, 1881. His work is to take foreign hides, salted as well as fresh, and after they have been soaked in water to transfer them to lime pits. He had had nothing to do with the hides after July 2nd because work was slack. On July 6th he noticed that there was a red itching swelling on his left cheek. He remembers also that it was moist and that he scratched it. The next day the place was much larger, and a quantity of "watery stuff" ran from it. Up to the 10th he felt well, had a good appetite, and slept as usual. The lump, however, got rapidly larger, and on that day (the fifth from its appearance) he had headache and loss of appetite. The next day he came up to the surgery and was admitted.

He was a healthy-looking, well-nourished man, and had always enjoyed good health. About the centre of the left cheek there was a swelling and eschar, of which the accompanying sketch (see Plate VIII), made at the time by Mr. Hurst, gives an accurate representation. It was a nearly circular area of slightly raised indurated tissue, $1\frac{1}{4}$ inches from above downward, $1\frac{1}{8}$ inches from side to side, and probably extending $\frac{1}{8}$ to $\frac{1}{4}$ inch down into the soft parts beneath the surface. In the middle it was dry and of a blackish-purple colour. At the sides it was covered with small vesicles, closely packed together, and containing straw-coloured serum. Around this was a red indurated surface from $\frac{1}{8}$ to $\frac{1}{4}$ inch across, ending somewhat abruptly in the healthier tissues, which were rather redder than normal. The area was nearly flat, higher by about $\frac{1}{20}$ inch at the edges than the centre, and raised from $\frac{1}{10}$ to $\frac{1}{8}$ inch above the adjacent skin. The submaxillary region and left side of the neck were red and swollen, and the œdema extended as low as the third rib

of that side. The cheek and eyelids were not swollen. I could detect no swelling of the lymphatic glands, but the reporter states that those in the posterior cervical region were enlarged and tender. The temperature was 101°, and the pulse quick.

Ether having been given I cut out the whole mass, making my incision a quarter of an inch clear of the margin. A considerable artery, probably the facial, and a corresponding vein bled freely. My dissection must have nearly reached the buccinator, for when I put my finger inside the cheek there appeared to be not more than one eighth to one sixth of an inch of soft parts covering it. I next applied a paste consisting of one part of chloride of zinc to three parts of flour to the cut surfaces at the edges of the wound, and finally I covered the parts with dry carbolic gauze.

In the evening his temperature had fallen to 100°, and it was never afterwards observed to rise above this level. The œdema rapidly disappeared, and the general health of the patient was at once restored. A large granulating wound, nearly two inches in diameter, was left, and when the slough caused by the chloride of zinc had separated, it was found that the caustic had opened Steno's duct. The salivary fistula thus formed somewhat retarded his complete recovery, but it was readily cured by the insertion of one end of a wire into the buccal orifice of the duct, while the other end was brought out into the mouth through the inner side of the duct, about a quarter of an inch higher up. The two ends were twisted together so as to form a wire seton in the last part of the duct, while the sides of the wound in the cheek were carefully adjusted with a button suture. I last saw him in December. His cheek was then completely healed, and he was but little disfigured by the scar, which measured one inch from above downwards, and half an inch from side to side.

A specimen was taken of the blood which flowed at the time of the operation, but owing perhaps to an imperfect

method of preparation, the results of the examination were negative. On making a section of the parts removed, I found that the blackened part in the centre extended very little into the true skin, but underneath it there was a congested patch of a dark purplish red colour in the corium and subcutaneous connective tissue. The indurated parts were at once placed in methylated spirit, and sections were subsequently made and stained after Koch's method by Dr. Charlewood Turner (see Plate IX). In these we discovered that the layers of the corium beneath the eschar contained abundance of large rod-like bodies, which were no doubt the *Bacilli anthracis*, as well as some collections of spore-like granules which appeared to have been formed by the breaking up of the bacilli. The accompanying drawings (see Plate IX, figs. 1, 2, 3) show the numbers and form of these bacilli, and Dr. Turner has kindly added a fuller description of their microscopic appearance.

Annexed is a table of the seventeen cases which have been admitted during the last nine years. They were all as well marked examples of malignant pustule as those which I have just related.

During the same period we have had under our care several cases of malignant facial carbuncle, but I have been careful to exclude these from my list. It will be seen that in all these cases the charbon has been observed upon an exposed part of the body. Fourteen out of the seventeen patients had been engaged in handling hides or fleeces, either as tanners or wharf labourers. One of them, however, had not touched hides for five weeks previously to his attack. But it is probable that some infectious particles had adhered to his clothes, from which he afterwards caught the disease. Of the three others in whom no history of infection could be obtained, one was a waterside labourer, and another was the wife of a man following the same employment, and all three lived in the neighbourhood of the wharves where the hides are landed. Possibly also in two of these cases a history of infection might

have been obtained, but they were the first which were observed, and the reports are in this respect imperfect. On the other hand, even if there should have been no direct contact, it is very possible that the poison may have been conveyed to them by flies which came from hides in their vicinity.

Our registrar, Mr. Symonds, has recently made some inquiries as to the source from which Case 1 was infected. The foreman of the warehouse from which the workman came said that the hides were chiefly from Cape Town, Bombay, Morocco, and Australia, and that they consisted of buffalo, ox, goat, and sheep skins. The workmen had no knowledge of the Van mohair, which was not long since the cause of an outbreak at Bradford. The Australian wool which the man had been handling was still in fleece. It was uncleaned, but the hide was dry and without odour. The foreman had been employed twelve years at this business, and had known only three men affected, viz. the present case, one admitted under my colleague, Mr. Lucas, in 1880, and another man who was treated in the London Hospital. There were several warehouses of the same kind in Tooley Street, but he had never heard of a similar disease at any of them. Neither he nor the workmen had any idea that the affection had any connection with the hides or wool. Mr. Symonds was also informed that a gentleman, the partner in a similar business, had died after a short illness from a spot in the back of the neck, like that which the labourers at the warehouses had had, but it had been attributed to the bite of a mosquito at Mortlake.

In the 'Daily News' of March 11th, 1882, there is the report of an inquest upon a similar case, in which a man, who had been working in a Bermondsey tanyard, died from spreading œdema accompanied by symptoms of blood-poisoning, which was attributed to the absorption of arsenic from the hides through an abrasion in the cheek. It will be seen that in several other instances recorded in my table, those affected stated that they had

lost fellow-workmen from the same disease. But I am not aware that it had in any case been attributed to poison derived from the hides.

The virulence of the poison is shown by the fact that some of those attacked had not handled the hides until they had undergone a long process of soakage in quicklime and water.

It will be seen that, in respect to age, our cases are pretty evenly distributed from that of eleven to forty-seven years. If we can draw any conclusion from so small a number of instances, it would be that old age and childhood are alike exempt, and that it occurs most frequently between puberty and middle age. Probably, however, this is the age of most of the workmen who are exposed to the infection.

According to Nicholai animals are most frequently attacked in August and September. This may account for the fact that the majority of our cases (twelve out of seventeen) were admitted in September and the four succeeding months. If we suppose that the hides were stripped from animals which had died from splenic fever in August and September, we might expect in the earlier part of the winter to meet with the disease in the men who had to handle the recently imported hides. Doubtless, however, many, if not most, of the hides came from countries where the time of prevalence of the disease in animals differs from that observed in Europe, so we should expect to find considerable variety in the seasons of the year in which the infection is likely to spread to men.

As yet I have not heard of any instances of internal infection, either by the alimentary or respiratory tracts, like those which have occurred at Bradford or those which have been described by Nicholai. I should expect, however, that now that our attention has been fully aroused to such a possibility, we shall find that some of our cases of severe inflammation of the lungs or intestines with septicæmic symptoms have been set up by this cause. There have also been, as far as I am aware, no cases of

malignant œdema without the formation of a primary central eschar, and I do not know that any have yet been observed in this country, unless those described by Dr. Budd, which seem to me to differ somewhat from typical cases of charbon, should be referred to this category. Out of 300 instances of external inoculation, Dr. Nicholai appears to have seen only two of this form. So it is not strange that in our limited number none should have yet occurred.

As a rule the diagnosis is easy. The raised indurated area with its central blackish depression surrounded by small vesicles can hardly be mistaken for any other affection. Generally, also, the painlessness of the swelling and the occupation of the patient will give some clue to the recognition of the disease. It has sometimes been confounded with malignant carbuncle of the face. But in carbuncle, even though there should be no evidence of central softening, there will usually be yellow spots of commencing suppuration upon the surface of the indurated mass. These spots, the severe pain, the absence of the characteristic eschar, and the evidence of implication of the adjacent veins, are sufficient for the discrimination of this disease.

I do not think that a simple poisoned wound could lead to any difficulty in diagnosis. In such a case there would usually be only one large and painful pustule, instead of numerous vesicles, in the centre of the inflamed area. The affection which I have found to resemble it more closely has been a primary chancre of the face, for here it is not uncommon to find a large scab upon a red indurated base, with considerable swelling of the soft parts and the contiguous glands. Probably, however, the history of the disease, its painfulness, and the slowness of its development, together with a closer inspection of the eschar, would enable us to form a correct diagnosis.

Four of our seventeen cases died, three from dyspnœa and the fourth with septicæmic symptoms. In one we were unable to make a post-mortem examination. In two

cases fibrinous exudations were found in the mucous membrane of the stomach and small intestines. The fourth had œdema glottidis. The veins were implicated in none of the cases. Two of the fatal cases were the first in my series. They were admitted in a moribund condition, and it was not thought necessary to excise the charbon. Of the fifteen cases in which excision was resorted to, twelve showed extension of the disease beyond the limits of the eschar, by inflammation of the soft parts, or swelling of the glands, and eight had well-marked constitutional symptoms. Yet all except two recovered rapidly. The fatal cases had a duration of 8, 9, 19, and 5 days respectively. In the three more rapid cases the charbon was upon the neck, and dyspnœa was a prominent symptom. This was shown to be due to œdema glottidis in the two cases which were examined after death.

With respect to the treatment, it is very important to remember that even after the swelling has extended to a considerable distance, and the adjacent glands have been affected, and after well-marked symptoms of blood poisoning have developed themselves, the patient may be restored to health by the removal of the indurated area of skin which was primarily attacked. As long as this remains, it acts as a focus from which fresh poisonous material is constantly being disseminated over the body; but after it has been removed, I presume that the system is enabled to eliminate that which has already reached the circulation, and so recovery takes place. In his work upon this disease, Bourgeois speaks of the treatment by excision as barbarous and obsolete, and he recommends the destruction of the eschar with caustic potash. In some of his cases, however, the disease appears to have spread notwithstanding the use of this remedy.

In the first case upon which I operated I began by using nitric acid, but I soon found that it ate too slowly through the indurated skin. So I gave up the use of the acid and proceeded to excise the whole of the inflamed

area. I subsequently applied chloride of zinc to the edges of the opening (see Table, p. 248).

This is the treatment which I have adopted on every other occasion, and always with success. But I am doubtful whether so severe a procedure is always necessary. Of course where the disease when unchecked is so fatal, it is better, as in a case of cancer, to err on the side of removing too much rather than too little of the affected tissue. As, however, on carefully examining the parts excised in my last case, I found that the bacilli, though abundant in the fibrous part of the skin and the sheaths of the hair follicles, were entirely absent from the subcutaneous fat in the deeper part of the section, I think that perhaps it was unnecessary to use the chloride of zinc in the subsequent treatment of the wound. On another occasion, if there was danger af the caustic reaching any important structure beneath, I shall be disposed to be content with the excision of the indurated skin, or I should take care to use afterwards some less destructive agent such as iodoform, perchloride of mercury, or a strong solution of carbolic acid.

Table of Seventeen Cases of Malignant

No.	Physician or surgeon.	Name.	Sex.	Age.	Occupation.	Admission.	Seat of charbon.	Days since attack.	Condition of adjacent parts.
1	Dr. Habershon	M.A.C.	F	11	—	Oct. 18, 1873	Under chin	6	Œdema of face; glands swollen
*2	Mr. Davies-Colley	E. M.	M	43	Waterside labourer	Nov. 22, 1873	Behind left angle of jaw	7	Hard œdema of neck
*3	„	J. F.	M	33	Tanyard labourer	Dec. 20, 1873	Tip of chin	6	No œdema around
*4	„	G. J.	M	27	Wharf labourer	April 7, 1875	Left cheek	7	Some swelling of glands under jaw
5	Mr. Howse	J. O.	M	47	Tanner	Sept. 4, 1877	Right cheek	15	Great swelling; glands enlarged
†6	Mr. Golding-Bird	D. D.	M	20	Dock labourer	Oct. 17, 1877	Right side of neck	3	Whole side of neck and shoulder much swollen
7	Mr. C. Forster	A. P.	M	17	Tanner	Jan. 18, 1878	Anterior surface of forearm	6	Glands enlarged
8	Mr. Howse	F. H.	M	18	Works among hides	Nov. 4, 1878	Under chin	3	Much brawny infiltration
9	Mr. Golding-Bird	J. E.	M	23	Works in tanyard	Nov. 13, 1878	Cheek	4	Swelling around; glands under chin enlarged
10	Mr. Bryant	J. R.	M	28	Hide cutter	Nov. 28, 1879	Forearm	5	Blush up to axilla

* Reported in 'British Medical Journal,' vol. i, 1878, p. 853.

Pustule treated at Guy's Hospital.

Constitutional symptoms.	Mode of infection.	Treatment.	Result.	Remarks, post-mortem appearances, &c.
Dyspnœa	—	Incisions	Died Oct. 20th	She lived in Horseleydown. P.M.—Œdema glottidis, and diphtheritic patches in the small intestine and vagina.
Dyspnœa; fever	—	Laryngotomy	Died Dec. 24th	Two years a total abstainer. P.M.—Œdema of larynx. No pus in veins.
None	Handling hides	Excision; chloride of zinc paste	Recovered	Had been six years a total abstainer.
None	Had been landing hides	Excision; chloride of zinc paste	Recovered	Told us that a similar affection had been fatal to some of his fellow workmen.
Very ill; vomiting; temp. 103°	—	Excision; chloride of zinc	Died Sept. 8th	Said it was frequent for tanners to be thus affected. P.M.—He was found to have fibrinous exudations in stomach and small intestines.
Temp. 103°; pulse 128	Five weeks ago unloaded dry skins	Excision; nitric acid	Recovered	Had an abscess of neck, and erysipelas during recovery. No bacilli in blood.
Sick; faint; no rest; temp. 101·2°	—	Excision	Recovered	—
Looks quite healthy and strong	—	Excision; perchloride of iron and caustic potash applied	Recovered	Nothing abnormal found in the blood.
Headache; shivering	—	Excision; carbolic and tannic acid	Recovered	—
None	Sheep and goat skins from Cape	Excision; chloride of zinc lotion	Recovered	No bacilli found in blood or serum. Two years ago a workman died of same disease, caught from similar hides.

† Reported in 'Guy's Hospital Reports, Series 3, vol. xxiii, p. 224.

No.	Physician or surgeon.	Name.	Sex.	Age.	Occupation.	Admission.	Seat of charbon.	Days since attack.	Condition of adjacent parts.
11	Mr. Lucas	D. W.	M	23	Wharf labourer	Oct. 10, 1880	Right cheek	5	Much swelling; eye closed; lip an inch thick; glands enlarged
12	„	S. C.	F	47	Domestic work	Jan. 4, 1881	Left cheek	11	Eye closed; glands under jaw enlarged
13	Mr. Jacobson	F. R.	M	31	Hide warehouse	April 16, 1881	Right lower eyelid	7	Upper eyelid; cheek swollen, also glands
14	„	F. F.	M	47	Stevedore	July 2, 1881	Back of fore-arm	4	—
15	Mr. Davies-Colley	T. W.	M	39	Tanner	July 11, 1881	Left cheek	6	Œdema to third rib
16	Mr. Golding-Bird	J. R.	M	27	Wharf labourer	Sept. 19, 1881	Cheek	3	—
17	Mr. Bryant	M. S.	M	22	Tanner	April 20, 1882	Right side of neck	3	Extensive œdema

Constitutional symptoms.	Mode of infection.	Treatment.	Result.	Remarks, post-mortem appearances, &c.
None on admission; two days before had fainted twice, and had had a sleepless night	Handling hides	Excision; cautery	Recovered	The day before he came in the swelling was still greater, so, perhaps, he was then recovering.
Very little	Husband unloads ships, but had not handled hides	Excision; cautery	Recovered	—
Sleeplessness; trembling	Australian fleeces	Excision; chloride of zinc	Recovered	Bacilli found in blood; fuller account in paper.
None	Australian wool	Excision	Recovered	—
Headache; loss of appetite; temp. 101°	Hides, but has not touched them since July 2nd	Excision; chloride of zinc	Recovered	Fuller account in paper.
No appetite; sick; limbs feel stiff	Hides, horns, &c.	Excision; galvanic cautery	Recovered	—
Dyspnœa; temp. 101·6°	Hides which had been soaked in lime	Excision	Died April 22nd	No P.M. He died from dyspnœa, remaining conscious the whole time. No bacilli in blood or serum.

Report on the Microscopical Examination of Sections from a portion of the Skin of the Cheek affected with Charbon, removed during life from a patient of Mr. Davies-Colley. By F. CHARLEWOOD TURNER, M.D., Assistant Physician to the London Hospital. (See Plate IX.)

THE specimens received for examination were portions of a piece of skin and subcutaneous adipose tissue of circular outline, divided by median crucial incisions.

The specimens were much thicker at their central than at their peripheral parts, the former being capped by a blackish eschar.

Thin sections were made parallel with the planes of crucial incision, thus showing one half of the complete median section of the portion of skin removed. The specimens were hardened in spirit only; in which they had been placed immediately after removal. The sections were made with the freezing microtome, stained in the methyl-aniline violet, and mounted in Canada balsam.

Inspection of the sections shows that the central thickening is due chiefly to swelling of the cutis. An early stage of this swelling of the cutis is seen in one of the sections shown, in a well-defined elevation of the Malpighian layer of the epidermis by serous infiltration into the most superficial part of the cutis.

In the other section there is seen at the border of the eschar a superficial vesicular formation, corresponding with the fringe of vesicles encircling the eschar described by Mr. Davies-Colley.

Immediately below the Malpighian layer at this part there is a defined area occupied by cloudy, granular, fibrino-serous exudation, studded with leucocytes, and containing numerous bacilli. The bacilli are aggregated in masses along the sheaths of the hair follicles, and along a well-defined boundary line between this area and the deeper tissue, which is also swelled and studded

with leucocytes, but presents a more delicate and clearer network, in which but few bacilli are to be seen, excepting along the sheaths of the hair follicles (see Plate IX, figs. 1 and 2).

The appearance is as though at this part there had occurred a separation between the cutis and the Malpighian layer of the epidermis above it, possibly from continued maceration of the tissue in the exuded serum, and that the sero-fibrinous exudation escaping into the space so formed had coagulated and undergone degenerative changes, affording conditions especially favorable to the development of the bacilli.

The bacilli are present in still greater abundance about the border of the eschar. The microscope shows that this eschar consists of the condensed and altered tissues of the Malpighian layer of the epidermis, and of the most superficial part of the cutis with the exudation coagulum in it. Along the deep surface of the eschar is a dense aggregation of leucocytes, and at the inner border of this numerous bacilli are visible. But they are most abundant about the margin of the eschar, and more especially just beneath the Malpighian layer of the epidermis. Dense aggregations of the bacilli are seen at several points; many of which appear to be formed in spaces in the exudation material. With them are many deeply-dyed, coarsely-granular masses (? zoogloœa). In places, as shown in fig. 3, the bacilli forming these clusters seem to be studded with sporules; some of the bacilli are broken up into short pieces; and rows of granules, similar to those composing the contiguous granular masses, appear as the relics of other bacilli.

In portions of exudation matter still adherent at the surface of the Malpighian layer at this part, with fragments of the cuticle, clusters of bacilli are here and there to be seen, and also deeply stained granular masses.

In the papular elevations of the cutis just beyond the border of the eschar a few bacilli are to be seen, beyond this there appear to be no important changes in the struc-

ture of the cutis, and no bacilli were observed. In the deeper part of the cutis in the central region but few bacilli could be seen, and in the adipose tissue, which is greatly swelled, especially at the borders of the section, where the fat cells are widely separated from each other, a careful search failed to afford evidence of the presence of bacilli.

Immediately below the eschar several vessels of considerable size are seen in section. In some of them bacilli are seen.

In the subcutaneous adipose tissue many arteries and veins of considerable size are included in the sections. Many of these were examined, but in none was any definite evidence of the presence of bacilli found. This tissue was the seat of much sero-fibrinous and corpuscular infiltration, especially in the centre of the affected area. There the exudation in many parts has a cloudy, opaque, granular appearance, and the fat cells are widely separated from each other. The whole tissue is studded with leucocytes, and they are massed in great number along the vessels, and around many of the hair follicles and of the sebaceous glands connected with them; several of these last are seen to be invaded by the corpuscular infiltration, and the characteristic appearance of their epithelial structure is lost to a greater or less extent. No bacilli were discovered in these glands.

The bacilli seen in these specimens exceed the dimensions mentioned by Bollinger in vol. iii of Ziemssen's 'Encyclopædia of Medicine.' When measured with the camera the full-sized bacilli were found to be from $\frac{1}{1400}$th to $\frac{1}{1600}$th of an inch in length, some were seen larger. According to Bollinger's observations their length varies from ·007 to ·012 of a millimètre, or from about $\frac{1}{3700}$th to $\frac{1}{2000}$th of an inch. The bacilli are straight or slightly curved; several are to be seen dividing into two parts by transverse fissure.

The variety of forms presented by the leucocytes and their nuclei in these specimens is remarkable. Many

of the nuclei present an irregular hour-glass form, as though arrested in the process of active amœboid movements, others have a horse-shoe shape with moniliform character, as if about to divide into several parts. The greater number of the corpuscles have two, three, or more nuclei.

The nuclei are surrounded by a cloudy mass of protoplasm, which was quite distinct by its fuller staining, when the specimens were first examined soon after being mounted, but which has since become indistinct from a partial diffusion of the pigment. No cell-wall could be distinguished around the protoplasmic masses forming the corpuscles.

DESCRIPTION OF PLATES VIII AND IX.

Case of Malignant Pustule (J. N. C. DAVIES-COLLEY, M.C.).

PLATE VIII.

CASE 2.—Thomas W—. Malignant pustule on the left cheek; the facial outline has been added to show its position.

PLATE IX.

CASE 2.—Microscopic sections of a portion of the skin of the cheek affected with charbon.

FIG. 1.—Part of a transverse median section at the margin of the eschar—(× 60).

a. Malpighian layer of epidermis.
b. Sheath of hair follicle.
c. Boundary between space immediately below the Malpighian layer in which the bacilli are abundant, and clearer space in which there are few.
d. Margin of eschar.
e. Dense aggregations of bacilli.
f. Deeply stained granular masses.

FIG. 2.—A part of the same section at *b*, highly magnified, showing clustering of bacilli along the sheath of the hair follicle (*b*), and along the boundary line (*c*) shown above—(× 700).

In this drawing many bacilli are represented with one extremity tailing off. They are all of uniform thickness throughout.

FIG. 3.—A cluster of bacilli studded with granules and breaking up into granular bodies resembling those forming the adjacent masses—(× 700).

Hurst, del

Mintern Bros. Chromo

Fig. 1.

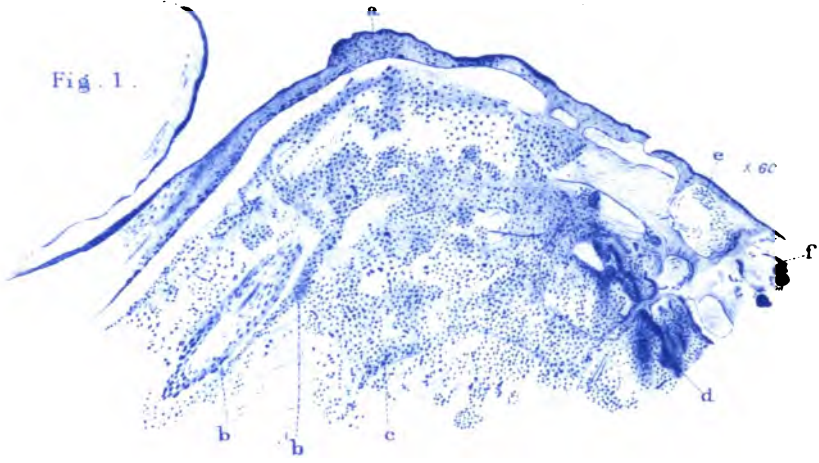
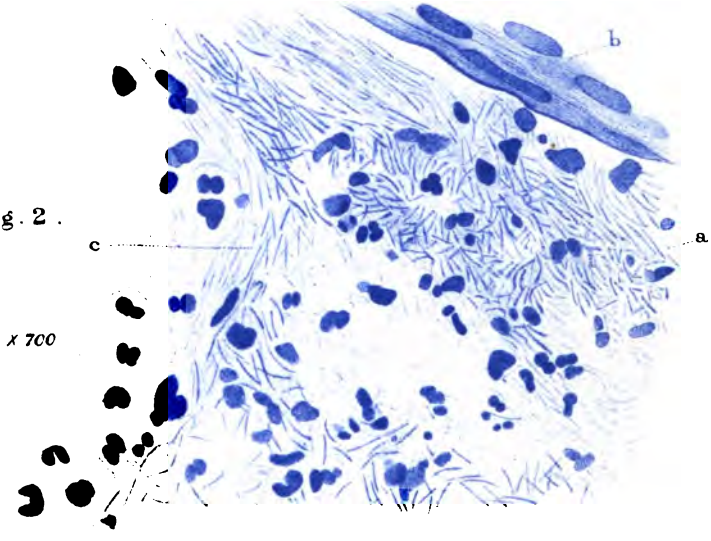
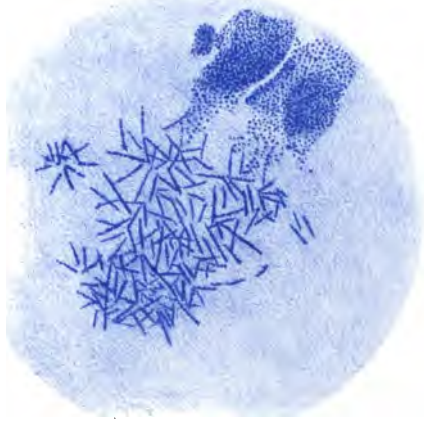


Fig. 2.



x 700

Fig. 3.



x 700

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